VOLUME 19 March, 1953 NUMBER 3

THE AMERICAN SURGEON

PUBLISHED MONTHLY BY
THE WILLIAMS & WILKINS COMPANY
BALTIMORE 2, MARYLAND

Subscription price $10.00 per volume. Single copies $1.00. Entered as second-class matter at the Post Office at Baltimore, Md, under the act of March 3, 1879.

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THE
AMERICAN
SURGEON

Vol. 19, No. 3 March, 1953

SURGICAL TREATMENT OF THROMBO-EMBOLIC DISEASE FOLLOWING FAULURE OF ANTICOAGULANT THERAPY

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Until recently there has been a great difference of opinion regarding the proper treatment of thrombo-embolic disease. The advocates of the surgical approach have been most enthusiastically in favor of the ligation of veins; the proponents of anticoagulant therapy have frequently made extravagant claims for their choice of treatment. In short, the question of correct treatment resolved itself into a controversy between the surgeon and the internist.

Over the years, as sufficiently large numbers of patients have been treated both surgically and medically to become statistically significant, a definite trend toward the use of anticoagulant therapy has developed. Both the surgical and medical approaches to this treacherous disease have certain dangers, contra-indications and advantages. It is extremely important, therefore, to select the method of treatment best suited to the individual. When it becomes apparent that one is not meeting with success with one method of treatment it is imperative that treatment be changed or modified promptly.

Until the ideal anticoagulant has been discovered and an accurate test has been devised to predict which patient will develop thrombosis or, as now seems unlikely, until the surgical approach has been demonstrated as being superior to medical treatment each case of thrombo-embolic disease must be considered individually.

The 4 cases to be reported were treated during the period from 1950 through 1952. They were selected only because they were consecutive patients who were seen during this period and in whom anticoagulants or other therapy was employed for varying periods of time before surgical therapy was eventually con-

Presented during the annual assembly of The Southwestern Surgical Congress, October 20–22, 1952, Dallas, Texas.

sidered. They were regarded as poor surgical risks at the time of operation, yet all of them recovered.

Case 1. A 28 year old salesman was admitted to St. Anthony's Hospital, Las Vegas, New Mexico on March 27, 1950 with the following history: One week before admission the patient was awakened during the night by severe substernal pain aggravated by inspiration. He arose and drove his car 70 miles to his home. He was forced to change a tire en route and felt very ill with pain in his chest and dyspnea. He felt better the following day but two days before admission to the hospital the pain recurred and he called his physician. The past history was important in that the patient had had a similar episode of chest pain a few months previously which disappeared in a few hours.

Course: On April 9, 1950, he developed hemoptysis and had obvious roentgenologic evidence of an infarct of the lower lobe of the right lung. Heparin was given for a week and he improved. He then developed increasing chest pain, dyspnea and hemoptysis. He continued to have fever and remissions and exacerbations of his symptoms until I examined him on May 11, 1950,—six weeks after the onset of his illness. Examination at this time revealed no clue as to the origin of the embolus except that there was slight swelling and tenderness of the calf of the left leg. The morning of the examination he developed fresh chest pain and hemoptysis. The inferior vena cava was ligated and he recovered uneventfully. He was dismissed from the hospital on May 24, 1950,—the fourteenth postoperative day and the fifty-seventh hospital day.

When last heard from, in August 1952, he was working full-time. He reported that he had had no further trouble, and no swelling of the ankles.

Comment: Part of the lack of success of anticoagulant therapy in this case was due to the fact that dicumarol could not be given because of inadequate facilities for laboratory control. It is obvious that the process was not controlled by heparin. If the operation had been done earlier it is probable that he would have been spared a very long and painful period of hospitalization.

Case 2. This 32 year old housewife was first admitted to St. Anthony's Hospital, Las Vegas, New Mexico, on June 15, 1951, with the following history: Two weeks after missing one menstrual period she developed vaginal bleeding and cramps. Dilatation and curettage was done. After the patient had left the hospital on June 16, the pathologist's report of decidual reaction without chorionic villi was received. She was next admitted on June 18, 1951, with a history of violent lower abdominal pain for six hours. On the following day, laparotomy was performed and a left tubal pregnancy with pelvic peritonitis was found. Left salpingectomy and appendicostomy were done.

Course: The patient's postoperative course was exceedingly stormy and was punctuated by epistaxis and bleeding from the abdominal wound. For many weeks her temperature ranged from 103 to 105 F. daily and she had multiple pulmonary emboli. She was dismissed on Aug. 15, 1951, but promptly developed another embolus. She was readmitted on September 11. Examination at this time revealed an acutely ill, emaciated woman with moderate respiratory distress. There were two draining sinuses in the lower midline scar. The erythrocyte count was 3,500,000 per cu.mm. and the hemoglobin was 68 per cent. There was moderate swelling and tenderness of the calf of the right leg and thigh.

Two days later the inferior vena cava was ligated. Technically, the operation was difficult, due to marked thickening and edema of the parietal peritoneum and fibrosis and edema around the vena cava. The patient made an uneventful recovery and was dismissed on the eleventh day following the ligation of the vena cava and 100 days after the onset of her illness.

When last examined on Oct. 1, 1952, she was in excellent health except for a hernia in the lower midline scar. She had minimal swelling of her ankles, which was not disabling.

Comment: This is the only patient in the series who did not receive anticoagulants, both

because of her hemorrhagic tendencies and the lack of laboratory control. It is possible that early femoral vein ligation would have controlled the process.

Case 3. This 32 year old housewife was admitted to the Espanola Hospital, Espanola, New Mexico, on Feb. 9, 1952, with a diagnosis of preeclamptic toxemia on the basis of blood pressure of 240/120, four plus albumen in the urine, and marked edema of the legs. The following day labor was induced and she had a normal delivery. She was dismissed considerably improved on the sixth day with blood pressure of 160/100. She was readmitted on February 18, with recurrent hypertension, severe back pain, and pyuria. Intravenous urogram showed no function of the left kidney. She was dismissed at her own request three days later, unimproved. Her next admission was on Feb. 23, 1952, because of marked swelling of both legs with extreme tenderness over the left femoral triangle. The pain in the leg was controlled by lumbar sympathetic blocks, after which dicumarol was started. Notwithstanding the fact that the patient's prothrombin time was adequately controlled, she developed increasing cyanosis, fever, and signs of extension of thrombosis in both lower extremities.

She was first examined by me on March 5, 1952, 10 days after her last admission to the hospital and nine days after anticoagulant therapy had been begun. It was thought that, in addition to multiple pulmonary infarcts, she had a fresh phlebothrombosis of the right lower extremity and thrombophlebitis of the left. After the prothrombin time had been controlled by large doses of vitamin K, the vena cava was ligated. She did well for three days and then developed massive swelling and pain in the entire right lower extremity. Sympathetic blocks were again done with improvement. On the fifteenth postoperative day, she developed severe pain in the chest, dyspnea, and cyanosis. Although a roentgenologic diagnosis of pneumonia was reported, she was given dicumarol because it was thought that she had an additional embolus. She gradually improved, and was dismissed on April 9, 1952, the thirty-third postoperative day, in fairly good condition.

When last examined on Oct. 10, 1952, she was in good condition, had had no further evidence of thrombosis or embolism, and had minimal swelling of both ankles which in no way interfered with her duties as a busy housewife.

Comment: This case is interesting in that the patient possibly had an embolus after vena cava ligation. It is possible that the source was the ovarian vein, and that she had a puerperal infection in addition to the thrombosis of the lower extremities. Such a clinical diagnosis was not made at the time, and the ligation was done through an extra-peritoneal approach. Another noteworthy fact is that the internist promptly faced the fact that notwithstanding adequate control with dicumarol, the patient was not improving, and he was ready to admit that other treatment should be instituted.

Case 4. This 31 year old white man was first seen by his physician on Aug. 19, 1951, with the following history: On July 15, 1951, while in another city, he developed tenderness in the calf of his left leg. He was given dicumarol for 10 days. Two weeks later he had a sudden onset of severe chest pain with dyspnea and cyanosis. He was treated for *pneumonia* for five days and sent home. On his return his physician made a diagnosis of pulmonary infarct and tromexane was given. He promptly improved. On Oct. 5, 1951, his chest was clear and he had returned to work. On Nov. 7, 1951, he was admitted to the hospital with a recurrence of his chest pain but with no other evidence of thrombosis. He was again given tromexane. He developed signs of progressive thrombosis of both lower extremities and became extremely ill, with dyspnes, fever, and signs of fluid in the left chest.

He was first examined by me on November 18, 11 days after admission to the hospital. At that time he was desperately ill with a temperature of 103 F., severe dyspnea, cyanosis, and massive pleural effusion. There were marked tenderness and swelling of the entire left lower extremity up to the inguinal ligament. There was moderate tenderness of the calf of the right leg without swelling. On Nov. 19, 1951, under local anesthesia, the left com-

mon femoral vein was ligated after three clots had been extracted from it. The clots were lying free in the lumen of the vein. The right superficial femoral vein was divided between ligatures. The patient had a stormy postoperative course with a long period of fever. At one time it was thought that he had a pulmonary abscess, but this disappeared and he was dismissed from the hospital on Dec. 19, 1951, on the thirty-first postoperative day.

Comment: It seems that the operation of choice in this patient would have been vena cava ligation. However, the patient's condition was so critical at the time of operation that it was thought he could not survive such an operation. Consequently, a compromise was agreed upon and apparently bilateral femoral vein ligation was justified in view of the results. The patient was last examined in September 1952, when he had minimal swelling of the left leg (the side in which the common femoral vein had been ligated), but was able to carry on his work as a contractor.

DISCUSSION

From a study of these brief case reports, it is obvious that they presented certain common problems. While the patient in case 2 was not given anticoagulants, it is presented as an example of the failure of the so-called *expectant treatment* of thrombo-embolic disease. The patient in case 1 received heparin in irregular doses and even then the clotting time of the blood was not continuously prolonged. Dicumarol was never administered in this first case because of inadequate facilities for determining the prothrombin time. The first 2 cases have in common the long delays, in case 1, 43 days and in case 2, 89 days, before their surgical treatment. From the outcome in these 2 cases it may be assumed that had surgical assistance been sought earlier the periods of hospitalization would have been considerably reduced.

Cases 3 and 4 shared the common factor that they were both adequately controlled with anticoagulants but notwithstanding this they developed further thrombosis and embolism. In case 3 the lack of control was recognized within eight days and an operation was done. Notwithstanding the operation, it is thought that this patient may have had another embolus. Case 4 apparently responded well to anticoagulants after the diagnosis had been made, and went through a considerable period of good health during which time he was given anticoagulants as an outpatient with careful laboratory control. When finally the patient came to operation, his condition was such that the proper operation, vena cava ligation, could not be done safely. It is extremely fortunate that he responded well to a procedure which was definitely a compromise.

Notwithstanding the present popularity of anticoagulants in the treatment of thrombo-embolic disease it is considered that there are at least three definite indications for surgical treatment:

1. When the use of anticoagulants cannot be properly controlled by adequate laboratory procedures.
2. In patients in whom there is a specific contraindication to the use of anticoagulants, such as liver damage or a recent operation in which hemorrhage is feared.
3. In those patients who have not responded to anticoagulants under so-called proper control. A plea is made for prompt recognition of the last class of patients, and the institution of appropriate surgical treatment.

SUMMARY

Four cases of thrombo-embolic disease are presented in which medical treatment failed but finally rseponded to surgical treatment.

Although it is generally considered that anticoagulant therapy is the method of choice in thrombo-embolic disease, there are certain contraindications to its use.

On the basis of the cases presented, it is thought that one of the most important contraindications to continuing anticoagulant therapy is in those cases in which it has obviously failed. A strong plea is made to recognize such cases early, and to institute a change in treatment promptly.

ACUTE HEAD INJURY: A REVIEW OF ONE THOUSAND CASES

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The rising incidence of injury by assault and vehicular accidents has broadened the problem of head injury. Since Merritt's[4] review of the literature in 1943, many others[1, 2, 3, 5, 6, 7, 8, 9, 10] have made contributions to this subject. To analyze the special issues of the general problem of head injury as they applied particularly to our own management, we studied 1,000 acute cases admitted to Hahnemann Hospital in the period from 1945 to 1950.

Approximately 40 per cent of all the patients seen in the emergency room of this hospital with the chief complaint of head injury were not admitted. Only those with histories or physical findings suspicious of cerebral involvement or indicating actual cerebral damage, as evidenced by loss of consciousness or other neurologic deficits, were hospitalized. We seldom saw the textbook picture of serious head injury as a single pathological entity. In many instances, we encountered multiple injuries, cranial and extracranial, or concomitant medical disease, *i.e.*, alcoholism, cerebrovascular accident, meningovascular syphilis, diabetes mellitus, cardiac disease, and malnutrition. The head injuries in our series were attributed to falls 30 per cent, vehicular accidents 30 per cent, assault 24 per cent, foreign bodies 5 per cent, and undetermined 11 per cent. Five hundred and seventy of the 1,000 hospitalized patients manifested signs of cerebral damage; 430 showed no cerebral involvement. We thought it important, as a basis for our analysis, to classify these head injuries as objectively as possible. This classification presented a practical clinical as well as a pathological approach to the patient (Table I).

Concussion was the only cerebral injury diagnosed in 442 or 77.5 per cent of 570 head injuries with cerebral involvement (Tables II and III). Three hundred and fourteen of these concussions occurred as single injuries; 128 occurred with skull fractures and were classified as multiple head injuries. There were 17 deaths in the group, however, each was complicated by severe, multiple extracranial injuries. The remaining 128 patients suffered more serious cerebral damage. Among them were 38 subdural, 11 epidural, and 2 combined subdural-epidural hemorrhages. Twenty-four of the subdural 63 per cent, 9 of the epidural 81.8 per cent, and both combined subdural-epidural hemorrhages 100 per cent, died. For these 51 serious acute cerebral lesions, there was an average mortality rate of 68.6 per cent.

The diagnosis of *fractured skull* was made in 219 of the 1,000 cases reviewed. Sixteen of these occurred among the 430 patients who had no apparent cerebral injury, an incidence in that group of 4 per cent; 203 occurred among the 570

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patients with apparent cerebral injury, an incidence of 36 per cent. There were 35 basal skull fractures among the 219 fractures, one of which was in the group with no cerebral involvement. Roentgenograms demonstrated the fracture in 75 per cent of the cases in which they were taken. When considering those frac-

TABLE I

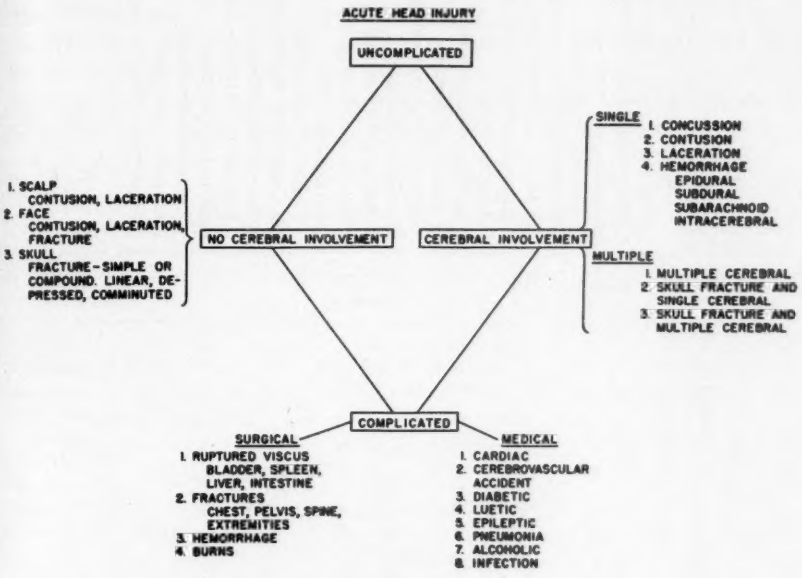

TABLE II

570 cases of acute head injury with cerebral involvement

Single Injury		Multiple Injury	
Concussion	314	Multiple cerebral	12
Contusion of the brain	18	Single cerebral and skull fracture	176
Laceration of the brain	6	Multiple cerebral and skull fracture	27
Hemorrhage			
Epidural	1		
Subdural	13		
Subarachnoid	2		
Intracerebral	1		
	355		215

tures which were not of the basal type, the roentgenologist was able to demonstrate the fracture in 85 per cent of the patients; the accuracy of roentgenologic diagnosis with respect to basal skull fractures dropped to 33⅓ per cent. In all, 78 of the 219 fractures or 35 per cent were diagnosed on a clinical basis. There

TABLE III

215 cases of multiple head injury

Multiple Cerebral Injury		Single Cerebral Injury and Skull Fracture		Multiple Cerebral Injury and Skull Fracture	
Diffuse cerebral	3	Concussion	128	Contusion and laceration of brain	1
Laceration of brain	2	Contusion of brain	21	Contusion and laceration of brain	7
Subdural hemorrhage				subdural hemorrhage	
Subarachnoid hemorrhage				subarachnoid hemorrhage	
Contusion of brain	1	Laceration of brain	10	Contusion and laceration of brain	6
Subdural hygroma				Subarachnoid hemorrhage	
Subarachnoid hemorrhage				Contusion of brain	2
Contusion of brain	1	Subdural hemorrhage	7	Subdural hemorrhage	
Subdural hemorrhage				Contusion of brain	1
Subarachnoid hemorrhage				Intracerebral hemorrhage	
Laceration of brain	1	Epidural hemorrhage	7	Contusion of brain	1
Subarachnoid hemorrhage				Subdural hemorrhage	
Contusion of brain	1	Subarachnoid hemorrhage	3	Epidural hemorrhage	
Laceration of brain				Laceration of brain	1
Subdural hemorrhage				Epidural hemorrhage	
Subarachnoid hemorrhage				Intracerebral hemorrhage	
Laceration of brain	3			Subdural hemorrhage	5
Subdural hygroma				Subarachnoid hemorrhage	
				Subdural hemorrhage	1
				Epidural hemorrhage	
				Epidural hemorrhage	2
				Subarachnoid hemorrhage	
	12		176		27

were 53 deaths among the 203 patients who sustained fractured skull with cerebral damage. All had severe cerebral involvement or multiple extracranial complications. In comparison, among the 367 patients with cerebral damage without skull fracture, there were but 21 mortalities. Therefore, in the individual case, a skull fracture may have serious prognostic significance, not only in relation to the severity of cerebral damage, but also with respect to complicating systemic injury or disease.

There were 196 fractures of bones other than skull among the 1,000 acute head injuries. Fractures of the jaw and nose accounted for 74, fractured ribs 40, pelvis 10, and vertebrae 2. The mortality rate among head cases complicated by rib or pelvic fractures was high, 35 per cent and 30 per cent respectively. Such

TABLE IV

Analysis of 74 mortalities among 570 cases of cerebral injury

Uncomplicated Head Injury		Complicated Head Injury	
Single head injury			
Concussion	0	Concussion	0
Contusion of brain	2	Contusion of brain	0
Laceration of brain	0	Laceration of brain	2
Hemorrhage		Hemorrhage	
Epidural	1	Epidural	0
Subdural	1	Subdural	4
Subarachnoid	0	Subarachnoid	2
Intracerebral	0	Intracerebral	1
Multiple head injury			
Single cerebral and skull fracture	5	Single cerebral and skull fracture	23
Multiple cerebral and skull fracture	8	Multiple cerebral and skull fracture	17
Multiple cerebral	2	Multiple cerebral	6
	19		55

injuries may not be recognized by the inexperienced because of attention to apparently severe superficial wounds of the head, or the inexperienced house physician may think that the cyanosis and dyspnea from a crushed chest are due to the head injury and consequently neglects the serious extracranial complicating condition.

There were 74 mortalities in this series of 1,000 cases, a mortality rate of 7.4 per cent. All of them occurred among the 570 head injuries with cerebral damage, 13.0 per cent of this group. Nineteen of these were uncomplicated head injuries; the other 55 were complicated by some medical-surgical disease (Tables IV and V).

Seventy-four per cent of our patients who died had head injuries complicated by other medical-surgical disease, the most frequent of which was the

TABLE V

Analysis of the complicating factors in 55 complicated head injury mortalities

Medical		Surgical		Both	
Prematurity	1	Multiple fractures	5	Congenital defects (newborn)	1
Atelectasis	1	Simple fractures	5	Rib fractures, pneumonia	1
Alcoholism	12	Rib fractures	5	Single fracture, cardiac failure	1
Illuminating gas intoxication	1	Multiple fractures and fractured pelvis	1	Fractured ribs, pelvis, atelectasis, lung abscess, cardiac failure	1
Subpleural hemorrahge	1	Multiple fractures and rib fractures	2		1
Tracheobronchitis	1	Fractured ribs and pelvis	2		
Pneumonia	5	Multiple fractures, hemothorax	1		
Pneumonia and meningitis	1	Rib fractures, hemopneumothorax	1		
Meningitis and atelectasis	1	Fractured ribs, contusion of pancreas, hemothorax, hemoperitoneum	1		
Epilepsy	1				
Aortic aneurysm	1				
Hypertension	2				
	28		23		4

respiratory complication. Serious respiratory difficulties were present or arose in 44 per cent of these patients, alcoholism was present in 22 per cent, fractures other than head and chest in another 22 per cent, meningitis in 4 per cent, and the remainder were problems of the newborn or cardiovascular disease.

Thirty of the 570 patients with cerebral involvement developed respiratory embarrassment to such a degree that asphyxia became an urgent and immediate problem. Each of these patients was brought to the emergency room with severe injuries and in deep coma or shock. Each had an obstructed airway due to blood, mucus, or tongue and some degree of cyanosis. Twenty required tracheostomy, 10 endotracheal suction. Oxygen was administered by tube; in addition, almost uniform therapy included whole blood, plasma, antibiotics, and fluid-electrolyte replacement. The degree of injury was so great that 24 or 80 per cent of these patients subsequently died; 4 within two hours, and 5 within 20 hours after admission. However all 6 who survived were in that group who had tracheostomy. Multiple bronchoscopies and endotracheal suction were tried with the cooperation of both the thoracic surgical and anesthesia departments. These methods often proved impractical and we eventually concluded that tracheostomy was best.

The one constant feature of all 570 head injuries with cerebral damage was a loss of consciousness at some time following the accident. In the 442 diagnosed as cerebral concussion, no other abnormal neurologic findings were present. As noted in Tables II and III, 128 patients were found to have more serious cerebral damage, as demonstrated by history, physical or laboratory examinations, and diagnosed accordingly. Of these 128 patients, 88 or 69 per cent showed abnormal neurologic signs other than coma. In the remaining 31 per cent, the recorded neurologic examination disclosed no significant abnormality and presumably did not reflect the true extent of the cerebral damage. Spinal tap, craniotomy, or necropsy revealed the actual degree of cerebral pathologic change. Apparently in a few instances, the record was incomplete; this fact notwithstanding, it can still be said that at least 25 per cent of the patients with severe injury to the brain showed no other signs than coma.

Sixty-five per cent of 43 cerebral contusions and 60 per cent of 20 cerebral lacerations presented neurologic manifestations other than a loss of consciousness. Seventy-four per cent of 38 subdural, 73 per cent of 11 epidural hemorrhages, and 100 per cent of 4 hygromas had abnormal neurologic findings. There were lateralizing pupillary changes in 11 patients; 6 occurred with unilateral hemorrhage or hygroma and lateralized to the ipsilateral side, the other 5 appeared in patients with bilateral lesions. The acute epidural, subdural hemorrhages, and subdural hygromas consistently showed the greatest number of lateralizing and localizing signs (Table VI).

Lumbar punctures were recorded in 172 instances; 70 were negative, 102 showed abnormal findings. In the latter, gross blood was demonstrable in 90, increased pressure in 51. In view of the nonspecific nature of these figures, the only conclusion justified is that lumbar puncture was a diagnostic aid in some cases, made a few patients more comfortable, and was of medicolegal importance

TABLE VI

Neurologic manifestations in 570 acute head injuries with cerebral damage

	Total No. of Cases	Coma	No. Cases with other Neuro. Signs	Reflex Change		Eye Changes				Paresis or Paralysis	Convulsive Movements	Decerebrate
				Hyperreact.	Hyporeact.	Nystagmus	Dilated Pupil		Papilledema			
							Ipsi	Contra				
Concussion	442	442	0									
Contusion of brain	43	43	28	10	18	1	(2 bilateral)		1	1	1	
Laceration of brain	20	20	12	5	5	2	(1 bilateral)		1	2		3 (3 died)
Epidural hemorrhage	11	11	8	5	2		1					2 (1 died)
Subdural hemorrhage	38	38	28	15	11	4	4	—	4	6	3	2 (2 died)
Epidural and subdural	2	2	1	1	1							
Subarachnoid hemorrhage	5	5	5	4	1					2		
Intracerebral hemorrhage	2	2	0									
Hygroma	4	4	4	2	1		(2 bilateral)		1	1		
Diffuse	3	3	2	1	1							1 (1 died)
	570	570	88	43	40	7	6 (5 bilateral)		7	12	4	8 (7 died)

in others. In 1 patient, it was judged that spinal taps were helpful therapeutically during a stormy postoperative course.

Exploratory cranial burr holes were done on 44 patients of whom 17 or 39 per cent eventually died. These 17 patients comprised 23 per cent of our total mortalities. Burr holes were done in 76 per cent of the cases of meningeal hemorrhage or hygroma. The high mortality rate in these patients suggests that either the burr holes were done too late, or the underlying cerebral injury or extracranial injuries were so severe as to be fatal injuries.

DISCUSSION

The increasing number of head injuries remains a public health and hospital administrative problem. The ratio of patients observed to the available hospital space and personnel is often excessive. In our hospital, it was not practical to admit every head injury brought to the emergency room. With our classification as a basis for judgment we relied on a supervised house staff to make the correct decision in each case. Did the percentage of preventable error justify such a policy? This question must be answered by each hospital faced with the same problem. Such a policy at this institution resulted in three serious complications during the period 1945 to 1950. One patient who was not admitted was reported to have died elsewhere, 1 was returned in deep coma with an epidural clot, and the other was brought back with meningitis. The latter 2 also subsequently died. These patients when first seen allegedly exhibited no real evidence of external injury and gave no certain history of unconsciousness. Therefore, we became convinced that if such a patient presents a history of a serious accident or hard blow, he should be hospitalized on suspicion. This holds true especially for alcoholic patients who should be kept under medical observation until they are mentally clear. On the other hand, there were 430 patients in our series who eventually proved to have no notable cerebral damage. These people at the time of admission were diagnostic problems; each was suspected of having cerebral injury and possibly serious complications. The true nature of their status became apparent only after careful observation for a period of at least 24 hours.

The classification used combines both a clinical and pathological approach to the acute head injury. It recognizes that a substantial number of head injuries are associated with other systemic injuries or concomitant medical-surgical diseases, and enables the house physician who first sees the case to take a more objective view of the patient.

A skull fracture alone may constitute but a minor injury. However, when it is realized that skull fractures are often associated with severe cerebral or extracranial injury, this fracture assumes serious prognostic significance. Roentgenograms of the skull should be taken some time during the patient's hospital stay. Generally speaking, these should not be made until after the period of shock has passed. However, the old admonition that taking roentgenograms in the seriously injured should be condemned, must be qualified. There were cases in whom accurate roentgenologic diagnosis was essential to the proper management, shock or not, especially when the chest or abdomen was involved.

The use of antibiotics in head injuries should be routine in those patients who bleed or leak cerebrospinal fluid from the ears, nose, or mouth, or show any signs of meningeal irritation. Generally, penicillin with intravenous sodium sulfadiazine as an important adjunct was the antibiotic of choice for routine cases. There were 2 patients with meningitis in our series, both of whom died notwithstanding vigorous therapy; however, each had fully developed meningitis together with severe pulmonary complications at the time of admission. Both had skull fractures with bleeding from the ears. These were the only patients with meningeal infection in a group of 73 cases with bleeding or leakage of cerebrospinal fluid from the ear, nose, or throat.

When judging the severity of cerebral damage, the observation of the presence or absence of coma and its degree was of paramount importance. In many cases of serious head injury, localizing and lateralizing signs did not appear. At least 25 per cent of head injuries with cerebral damage showed no manifestations other than coma. Therefore, it is imperative that all personnel, both professional and nonprofessional, be trained to note any changes in degree of consciousness. The indications for exploratory burr holes were as follows:

1. Coma which was persistent, deepening, or intermittent.
2. Progressive deterioration, slow or rapid.
3. Persistent or progressive abnormal neurologic signs.
4. Prolonged stationary course.
5. Patient, admitted in critical condition, whose history and clinical course could not be accurately determined and in whom there was a high index of suspicion.

If there was any doubt, it became our policy to operate. We had no cause to regret this aggressive policy. The patients tolerated the relatively simple operative procedure well. In acute intracranial hemorrhages the prognosis is poor; even so, those who can be saved or helped should be operated upon. Operative intervention was also indicated in those patients with compound skull fractures requiring debridement, or those cases of depressed fracture requiring elevation of the depressed bone segment.

Respiratory embarrassment was a most serious complicating factor. Tracheostomy was indicated in patients with manifest respiratory obstruction or prolonged coma. We usually preferred tracheostomy to endotracheal suction because:

1. It was a well tolerated, simple, single procedure.
2. It was direct and definitive.
3. Less experienced ward help could take over aspiration and management of the oxygen catheter.
4. Alcoholic, intractable, and severely injured patients were most easily managed in this manner.

The patient should be closely observed for the development of respiratory complications. If such complications occur, action should be prompt. There is a decided inertia on the part of most observers in this respect. If there is any doubt, we believe it is best to do a tracheostomy as early as possible.

In our series, as well as others, the high mortality rate of 40 to 60 per cent for severe head injuries is primarily due to either extensive, multiple cerebral damage, usually contusions and lacerations of the brain, or serious extracranial complicating factors (Table VII). Death rates in reported series have little significance unless they are based on a classification according to severity of injury and complications. The overall mortality in most institutions has been reduced from 15 or 20 per cent to less than 10 per cent. This is because a larger number of minor injuries and noncerebral cases now being admitted, give weight to the more favorable result. The group suffering severe injuries, when analyzed in all reported series, still presents a gloomy outlook. This group of seriously injured people comprises 10 to 15 per cent of all acute head injuries. The patients in this

TABLE VII

Mortality of acute head injury

Type of Injury	Number of Cases	Mortality Rate, %
Acute head injury, overall	1000	7.4
Acute head injury without cerebral involvement	430	0.0
Acute head injury with cerebral involvement	570	13.0
Single injury	355	3.7
Concussion	314	0.0
Contusion of the brain	18	11.1
Laceration of the brain	6	33.3
Hemorrhage	17	53.0
Multiple injury	215	28.4
Single cerebral and skull fracture	176	15.9
Multiple cerebral	12	66.7
Multiple cerebral and skull fracture	27	92.6
Cerebral injury with skull fracture	203	26.1
Cerebral injury without skull fracture	367	5.7
Acute head injury, complicating rib fractures	40	35.0
Acute head injury, complicating pelvis fracture	10	30.0
Acute head injury, exploratory burr holes	44	39.0
Acute head injury, tracheotomy	20	60.0

category should be recognized almost from the beginning. It is true that in some patients the injuries are so severe that they exclude any reasonable hope for recovery. Still, we must mobilize all our resources for this type of patient. Here is where we must direct our best efforts; here, all methods and results stand the test of critical appraisal; here there is room for improvement.

SUMMARY

1. One thousand cases of acute head injury are reviewed.

2. A broader classification of acute head injury is presented which emphasizes those factors which most directly contribute to the seriousness of the injury.

3. Mortality rates of acute head injuries in relation to these factors are presented.

4. The special problems, encountered and discussed are: selective hospitalization of head injuries; the use of roentgenography in the complete diagnosis of these cases; significance of neurologic signs and coma; spinal tap; concomitant systemic injury and associated medical disease; factors contributing to mortality; indications for tracheostomy; and indications for exploratory craniotomy.

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INTRACEREBRAL HEMATOMA

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Intracranial hematoma, also known as subcortical hematoma, has been a subject of increasing interest in recent years. Many authors including Pilcher,[13] Craig and Adson,[2] and Hamby[9] have delineated the clinicopathologic aspects quite clearly, and their contributions have alerted physicians interested in diseases of the central nervous system to its existence. Increasing recognition and the surgical relief of this entity have saved many persons who were otherwise doomed to die.

In various reports this disease complex has been divided into two primary categories:

1. Spontaneous.
2. Traumatic.

In this discussion a similar classification will be used and several case reports will be presented exemplifying each category.

Incidence. There are two types of traumatic subcortical hemorrhage:

1. Petechial.
2. The more massive collections which form the basis of this report.

According to Courville,[3] gross intracerebral hematomas are not common lesions, being found in about 8 per cent of cases of craniocerebral trauma.

Age. It has been suggested by Grant and Austin[7] that the traumatic cases tend to occur in the younger age groups. An analysis of several series of cases shows very little difference in the age distribution between the traumatic and spontaneous varieties.

Sex. There may be a greater incidence of this type of lesion in men because they are more often exposed to trauma by virtue of their being engaged in the more hazardous occupations and their naturally more aggressive behavior.

Location. Regardless of the etiology of the hemorrhage, whether spontaneous or traumatic, the most favored site of the bleeding is the temporal lobes. Grant and Austin[17] state that on the average approximately one-third to one-half of all intracerebral hemorrhages are located in the temporal lobe.

Predisposing Factors. Numerous hypotheses have been presented accounting for the formation of this type of hemorrhage. Evans and Sheinker[4] have proposed a logical explanation for this lesion. It has been their suggestion that it may follow trauma with resultant bleeding due to vasoparalysis, vasothrombosis and associated secondary hemorrhage. The accumulation of blood may also be secondary to the tearing of fairly large vessels. A third cause may be a coalescing hemorrhage composed of multiple small, bleeding vessels associated with a massive contusion.

Clinical Features. Usually intracerebral hematoma is not suspected before

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operation. One operates because there is evidence of a space occupying intracranial lesion, and since statistically acute subdural or extradural bleeding (particularly the former) are the most frequent complications of head trauma, exploration is usually done with the expectation of encountering one or the other. It is easy to confuse this entity with the more frequently encountered operable lesions, and the symptoms and signs are very often indistinguishable from those found in the extradural or subdural collections of blood. In general the following features may be found in this condition. In those rare instances in which the interval between the injury and the onset of the bleeding is prolonged, one may find a picture of the so-called delayed traumatic apoplexy. There have been examples of this occurring as long as several weeks and even months after an accident. The pyramidal system seldom escapes, there being in most cases some motor involvement varying from a hemiparesis to hemiplegia. Papilledema is practically always present and may be severe enough in the chronic cases to evoke concern over possible permanent visual impairment. Pupillary changes are not unusual but are clinically of little help in localization.

Examination of the cerebrospinal fluid reveals, in most instances, an elevation of pressure, usually gross and almost invariably microscopic evidence of bleeding. The degree of xanthrochromia is wholly dependent upon the duration of the lesion. Headaches and convulsions are frequently encountered.

Treatment. Therapy, of course, is directed toward evacuation of the clot. Since, in most instances, the presence of the hematoma is not suspected, treatment in the individual case will consist of exploratory trephination in the expectation of finding and removing an epidural or subdural hemorrhage. If neither of these conditions is encountered then one must be prepared to determine if there is an operable lesion present. This may entail the use of more complicated procedures such as ventriculography or cerebral angiography. In any event one must not be content to assume that the increase in intracranial pressure is due solely to cerebral edema and minor hemorrhages that cannot be evacuated. Although the incidence of gross traumatic clots is not very high, one must bear in mind that this lesion may occur as frequently as extradural hemorrhages of surgical proportions. As has been noted above, Courville[3] found in his series of cases an incidence of 8 per cent, whereas extradural bleeding has been found in from 2 to 7 per cent of all craniocerebral injuries.[8, 10] Certainly one would feel remiss in overlooking an extradural hemorrhage, since there has been so much attention drawn to this particular complication of head injuries. The prognosis following the successful treatment of the condition under discussion is equally as good as in the other lesions just mentioned and this fact alone warrants every effort to determine its presence or absence. Once the diagnosis is confirmed, evacuation may be accomplished by either aspiration through a trephine opening or by the more complicated procedure of craniotomy, depending on whether the hematoma is wholly liquid or consists of large, partly-organized clots.

The following 3 case reports are examples of the traumatic variety.

CASE REPORTS

Case 1. This patient was a 22 year old Negro man admitted to the hospital on July 25, 1950. He was hit on the head with a rock on July 22, three days before admission. He was not knocked unconscious. On the following day he could not be aroused from a deep stupor and continued in an unconscious state until he was admitted to the hospital.

On admission his blood pressure was 116/75, pulse rate was 54, respirations were 18 and temperature was 99.4 F. When first seen he was totally unresponsive, showing no reaction even to very painful stimuli. Examination of the head revealed no external evidence of trauma. The pupils were equal and reacted well to light. No papilledema was noted. The neck was rigid. The tendon reflexes were inactive. There was a normal response to plantar stimulation bilaterally. The cremasteric and cutaneous abdominal reflexes were bilaterally active and equal. Skull roentgenograms were normal. The cerebrospinal fluid was reported to be grossly bloody and xanthrochromic. No mention was made of the pressure.

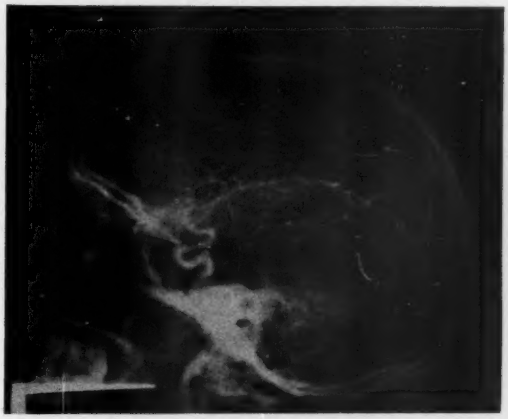

FIG. 1. In this cerebral arteriogram the Sylvian group of vessels is displaced upward suggesting a lesion of the left temporal lobe.

On the following day the right pupil was slightly larger than the left. He still responded very poorly. He was very drowsy and moved his extremities very sluggishly. Later that same day another spinal puncture was done. The initial pressure was 440 mm. of water and after the removal of 10 cc. of spinal fluid the final pressure was 220 mm. The fluid was still grossly bloody. On this date biparietal burr holes were made and nothing abnormal was found. Later, following operation he appeared slightly more responsive but not oriented. It was noted that he had a right facial palsy and also a left sixth cranial nerve weakness.

On August 2, a note was made that there was no papilledema. A left open internal carotid angiogram was done on Aug. 15, 1950 (fig. 1). On the day following this procedure he was quite well oriented. A large subcortical hematoma was subsequently found in the left temporal lobe and removed. His postoperative course was satisfactory with complete recovery.

Case 2. The next patient was a 42 year old white woman admitted to the hospital on the evening of May 1, 1949. She was struck by an automobile at about 8 p.m. and brought to the hospital in a conscious condition. At the admission desk she stated that "she didn't know what hit her." Skull roentgenograms revealed a linear fracture in the left occipital region. While in the clinic she became semicomatose and on admission to the ward lapsed

into deep stupor. The blood pressure was 180/90, the pulse rate 120, and the temperature 99.2 F.

Examination revealed a completely comatose woman who was having Cheyne-Stokes respirations. A subgaleal hematoma was found overlying the left parietal region, and a small laceration was noted in the right supra-orbital area. The ear drums were negative. The pupils were miotic but reacted slightly to light. The neck was not rigid. A flaccid paralysis of both the upper and lower extremities was found. The fundi were normal.

Later at 2:00 a.m. on May 2 her respirations were quite labored. The blood pressure was 200/120. The pulse rate was 121 and the respirations 40 per minute. The left pupil was slightly larger than the right. At 7:00 a.m. on May 2, she suddenly ceased breathing while being prepared for craniotomy. Artificial respiration was initiated and an endotracheal tube was inserted. Bilateral temporal burr holes revealed no extra or subdural hematoma. The brain was noted to be tense with no visible pulsations. The right temporal horn was cannulated and blood-tinged fluid was aspirated. The wounds were closed. No blood pressure or pulse was found at the close of operation. Later at 3:20 p.m. the blood

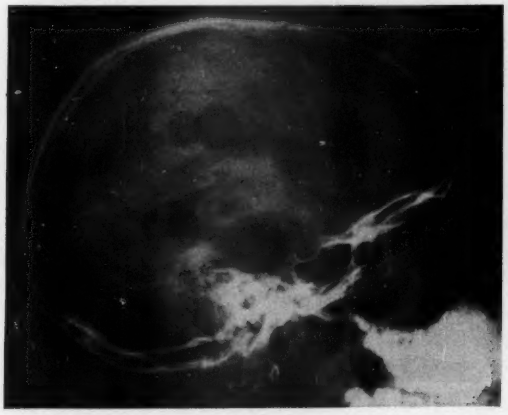

FIG. 2. This ventriculogram shows depression of the midportion of the left lateral ventricle, indicating a mass in the superior portion of the midparietal area.

pressure was 130. There was still no spontaneous breathing, but the patient would withdraw both legs on painful stimulation. There were no arm or face movements. On May 3 at 2:30 a.m., the blood pressure was 60 and at 11:00 a.m. she died. Autopsy revealed a cerebellar pressure cone. There were numerous small petechial hemorrhages throughout the brain. In addition there was a massive hemorrhage involving the left cerebellar hemisphere.

There have been few reports in the literature dealing with intracerebellar hematoma.[14] Most of these lesions occur above the tentorium and if there are no signs suggestive of posterior fossa difficulty the diagnosis is exceedingly difficult if not impossible without empirically placing burr holes over the cerebellar hemispheres in the hope of encountering such a complication. In the several instances of successfully treating a hematoma in this location recorded, there have been sufficient signs to indicate its presence. In this particular case, there is reason to doubt if recovery would have occurred even if the lesion had been recognized and evacuated, in view of the multiple petechial hemorrhages noted elsewhere.

Case 3. The next patient was a 10 year old boy who was admitted to the hospital on Dec. 13, 1951. The family gave a history of the boy falling down a six foot embankment the night before while playing. He was unconscious for several hours and then aroused and

was apparently quite well oriented. During the night it was noted by the family that he had definite weakness of the right upper extremity and almost complete paralysis of the right lower extremity.

On admission the boy was alert and quite responsive, complaining of pain in his neck and head. He was lying quietly in bed with chin pointed toward his left shoulder and resisted any attempt to straighten his neck or head. There was paresis of the right upper extremity and complete paralysis of the right lower extremity. The deep reflexes of the right arm were active and similar to those of the left arm. The deep reflexes of the right leg were hyperactive as compared with those of the left and there was an unsustained ankle clonus on the right. There was a definite extensor response to plantar stimulation on the right and a normal plantar response on the left. Funduscopic examination revealed no abnormalities. The cranial nerves were grossly normal. The ear drums were normal in appearance. The blood pressure was 124/74, the pulse rate was 98, the respirations were 22 and the temperature was 98.6 F. Roentgenograms were quite normal. Roentgenograms of the cervical spine were within normal limits except for straightening of the spine in the lateral view suggestive of muscle spasm.

On December 15, after being in halter traction for 24 hours with complete relief of his cervical muscle spasm but with no improvement in his neurologic picture, bilateral temporal burr holes were made and no evidence of an extra or subdural hemorrhage was found. On December 24, ventriculography was done using frontal burr holes and the roentgenograms revealed a moderate depression of the roof of the mid-third of the left lateral ventricle consistent with a left parietal space-occupying lesion (fig. 2). On December 27 a burr hole was placed close to the midline in the mid-parietal area and a large intracerebral hematoma, containing at least 30 to 40 cc. of old blood and clots, was evacuated after the burr hole was enlarged to facilitate removal.

Subsequently his course was essentially uneventful and he was discharged from the hospital on Jan 16, 1952. At the time of discharge he still had some residual weakness of the right lower and upper extremities but there was beginning improvement in his neurologic status.

SPONTANEOUS INTRACEREBRAL HEMATOMA

Incidence. In Courville's[3] series of 40,000 autopsies there were 1,487 cases of hemorrhage into the substance of the brain, or an incidence of about 4 per cent. However, the number of surgically resectable and removable lesions may be smaller than this and exact figures are not readily available.

Age. As noted earlier in this report the average age is apparently very little different in this group as compared with the traumatic variety. Hamby[9] reported an average age of 41.8 years in the spontaneous category as compared with an average age of 41.3 years as reported by Browder and Turney[1] in their series of traumatic cases.

Location. As in the traumatic cases the site of predilection is the temporal lobes.

Sex. There is apparently no great sex difference.

Predisposing Factors. Chronic diseases, notably arteriosclerosis and syphilis are the most common causes of this type of subcortical hemorrhage. Matas in 1938[12] claimed that 80 per cent of the deaths caused by cerebral hemorrhage were due to rupture of miliary aneurysms of the cerebral ganglionic branches of the circle of Willis. These aneurysms may be due to congenital defects, or to degeneration of the arterial walls associated with chronic arterial hypertension and arteriosclerosis. Globus and Strauss in 1927[6] believed that the concept of miliary

aneurysm rupture did not explain all the hemorrhages, but hemorrhages often result in areas of previous brain softening. This presupposes an antecedent episode of interruption of arterial blood flow, either by thrombotic, atheromatous or embolic occlusion, by buckling and kinking of dilated senile arteries within the internal carotid canal or by direct mechanical compression. Hemorrhage may result when the flow of arterial blood is maintained through exposed vessels in such areas of encephalomalacia leading to rupture of the softened arterioles within tissue of greatly reduced tensile strength.

Arteriosclerosis and hypertension were the essential causes of gross hemorrhage in 63 per cent of Courville's[3] cases. Seven per cent were due to embolism; 5 per cent were due to trauma, (the major portion of these occurring in association with birth injury); 3 per cent followed ruptured aneurysms; blood dyscrasias accounted for 2.7 per cent, and syphilis was the cause in 2 per cent. There were other causes also, but these were primarily secondary effects of systemic diseases, both metabolic and infectious.

Recent work by Odum[11] and his associates at Duke University place a good deal of stress on the role of small angiomatous malformations in the formation of these gross effusions in otherwise unexplained hemorrhages. Fincher[5] has also had several examples of gross collections of blood deep within the cerebral hemisphere in association with latent metastasizing cerebral melanomas.

Clinical Features. If the onset is abrupt without any history of antecedent trauma, one will usually suspect some form of intracranial vascular insult. In the majority of cases this will be the prevailing situation. In a few instances, however, the course may be quite prolonged, and the diagnosis in doubt, the presenting features suggesting only an intracranial space-occupying lesion, most probably a tumor. There is no need to enter into a detailed discussion in this report of the differential diagnosis of brain tumors. However, it might be well to note that in hemorrhagic lesions xanthrochromia of the cerebrospinal fluid is frequent whereas in brain tumors this is a relatively rare finding. In most other respects the clinical picture may be very similar.

No accurate figures of the relative incidence of spontaneous subarachnoid hemorrhage and surgically removable, gross, non-traumatic collections of blood within the substance of the brain are available, but one obtains the general impression that spontaneous subarachnoid hemorrhage is much more frequent. However, at times it may be quite impossible to differentiate between the two lesions. The onset may be as apoplectic and the clinical course can be quite similar. The spinal fluid findings also may be identical with elevation of pressure and grossly blood-tinged fluid. Only by specialized technics may the two conditions be differentiated.

The following case histories illustrate the non-traumatic form of hemorrhage within the substance of the brain.

CASE REPORTS

Case 1. The first example is a 31 year old man admitted to the hospital on Feb. 6, 1951. On the evening before admission, upon coming home from work he complained of a mild headache. He played with his children for a little while and then several hours later sud-

denely lapsed into deep coma, and had multiple clonic and tonic convulsions. No previous history of any type of illness was elicited except for occasional mild headaches and the possibility that he may have been treated in the past for mild hypertension.

When first seen the patient was in deep coma and could not be aroused even with very painful stimuli. He was in active extensor rigidity with mild intermittent clonic movements of the lower extremities and opisthotonous. The corneal reflexes were absent. The deep tendon reflexes were hyperactive throughout with bilaterally positive Hoffman and Babinski signs. No abdominal reflexes could be elicited. The pupils were miotic and did not react to light. On inspection of the fundi it was noted that the optic discs were definitely blurred at the nasal and superior margins and there was a flame-shaped hemorrhage at the superior margin of the left optic disc.

The blood pressure was 140/80; the pulse rate was 70; the respirations were 20 and the temperature 98.3 F. Cerebrospinal fluid examination revealed a pressure of 300 mm. of water. The fluid was clear and colorless with a red cell count on microscopic examination of 492 per cu. mm.

The patient died several hours later and at autopsy a large subcortical hematoma in the region of the left temporal lobe was found. No obvious source for the bleeding was found.

Case 2. This patient was a 28 year old woman admitted to the hospital on July 20, 1951. The family gave a history of having found the patient in deep coma and incontinent of urine on the floor of the bathroom on the morning of July 19. Some hours afterward she became quite alert and well oriented but complained bitterly of bitemporal headache. She was four weeks postpartum. The blood pressure was 140/100; the pulse rate was 70, and the temperature was 100 F.

At the time of examination, which was a day after the above described episode, the patient was well oriented as to time and place. However, there was definitely listlessness on her part when asked to perform certain tests during the examination. Muscular strength was physiologic throughout. The deep reflexes were active and equal bilaterally. The cutaneous abdominal reflexes could not be elicited. There was no incoordination noted on the right. However, there was some incoordination found on the finger to nose test on the left. Examination of the fundi revealed blurring of the nasal margins bilaterally. The remainder of the cranial nerves were grossly normal and no gross visual field defects were found. All modalities of sensation were within normal limits. Kernig's sign was mildly positive on both sides. Spinal fluid examination had been done and the fluid was recorded as being grossly bloody but no measurement of the pressure was mentioned.

The patient was diagnosed as having had a spontaneous subarachnoid hemorrhage and arrangements were made for arteriography. However, on the evening of July 21, she suddenly became comatose and died within a few minutes.

Autopsy revealed a large subcortical hemorrhage in the depths of the right cerebral hemisphere in the region of the basal ganglia with evidence of recent rupture into the right lateral ventricle, and massive hemorrhage throughout the ventricular system (fig. 3).

Case 3. The next patient was a 52 year old white man who was admitted to the hospital on Jan. 3, 1949. On January 1, two days before admission, he was in a fight and was struck on the head several times with a lead pipe. He was reportedly unconscious for one hour and then returned home the same evening and appeared to be normal. On the morning of admission he arose from bed; stated that he did not feel well and would not eat breakfast. A little later he fell on the sidewalk, and complained of sudden loss of function of the right arm and leg. About 10:00 a.m. he developed generalized tremors, and stated that he was having a chill. A half hour later he went to bed and shortly thereafter, when he tried to rise, he was unable to do so because of a right hemiplegia. At 11:00 a.m. he had a generalized convulsion with the sudden onset of coma which persisted. A review of his past history revealed that he had been under treatment for hypertension for the past eight years. Eight years before he had had a stroke with temporary paralysis of one side of his body (which

side was not known by the informant) with full return of function within three weeks. The blood pressure was 240/140; the pulse rate was 90; respirations 30 and the temperature 100.8 F.

Examination revealed a deeply comatose man appearing to be his stated age who with painful stimuli moved only his left side. There were numerous bruises on the forehead, right cheek, right neck, and manubrium of the sternum. The aural canals were negative. The left pupil was moderately dilated (about twice the size of the right). Both pupils reacted slightly to light. There was a right central type of facial paralysis. Examination of the fundi revealed a bilateral choke. No stiffness of the neck was noted. There was a complete paralysis of the right upper and lower extremities. The deep reflexes on the right were hypoactive as compared with the left with absent abdominal reflexes. There was an extensor response to plantar stimulation bilaterally.

On the same date cerebrospinal fluid examination revealed an initial pressure of 570 mm. of water. The fluid was grossly bloody. The non-protein nitrogen was 60 mg. per 100 cc. Urinalysis was negative. On Feb. 3, 1949, skull roentgenograms revealed no fracture of the calvarium, but there was a comminuted fracture of the right malar bone with minimal de-

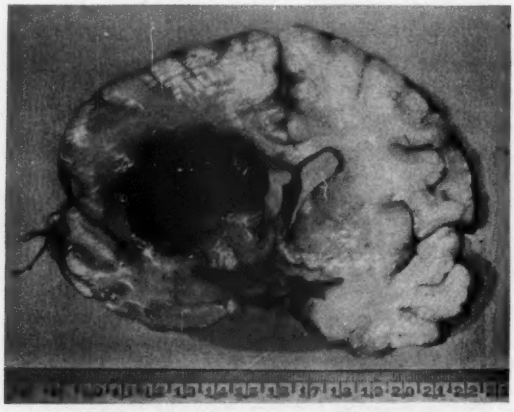

FIG. 3. A large blood clot is present in the depths of the right cerebral hemisphere, and at one point has ruptured into the right lateral ventricle.

pression. On February 5, bilateral frontal and bilateral parietal burr holes were made. The brain appeared tense and no pulsations were noted. No extra or subdural hematoma was encountered. On February 6 the patient died. Autopsy revealed a very large intracerebral hematoma about 7 cm. in diameter in the region of the left internal capsule extending longitudinally from the tip of the left lateral ventricle posteriorly to the region of the left cerebral peduncle. The left lateral ventricle was flattened out toward the midline with no point of rupture.

This is an equivocal case and one in which it would be difficult to determine if the injury caused the hemorrhage. It is interesting to note that without any hesitation the pathologist stated that he believed the hemorrhage was secondary to the hypertension and that the trauma was incidental.

SUMMARY AND CONCLUSIONS

A discussion of intracerebral hematomas has been presented with an analysis of the relative incidence, pathogenesis, clinical features and treatment in both the spontaneous and traumatic varieties. Six case histories have been reviewed exemplifying each of the two categories.

In conclusion there are two important points to consider.

1. In instances of craniocerebral trauma with a strong clinical impression of an intracranial space-occupying lesion, every effort should be made to rule out the existence of an intracerebral hematoma, if the more obvious causes such as an extra or subdural hemorrhage have been excluded. It may be necessary to resort to ventriculography or cerebral arteriography to prove or disprove the presence of such a lesion, but one should not hesitate to employ these measures if indicated.

2. There is still some discussion as to the optimum time to perform arteriography in cases of suspected spontaneous subarachnoid hemorrhage. Some authors boldly advise immediate use of this procedure as soon as the diagnosis is entertained. Others believe that there is a definite risk involved, with danger of spilling the dye into the basal cisterns and also it is believed that if several days are allowed to elapse that intracranial attack on the source of the bleeding may be technically much easier. Probably there is, as usual, a middle ground with the always desirable feature of case individualization. But one should bear in mind, if delay in resorting to angiography is decided upon, that, in the rare instance, one of these surgically resectable and curable lesions may be overlooked. Clinically it may prove impossible to differentiate between a gross intracerebral hemorrhagic effusion and a subarachnoid hemorrhage resulting from a ruptured intracranial aneurysm.

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TRACHEOSTOMY: ITS ROLE IN POSTOPERATIVE AND POST-TRAUMATIC CARE

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Recently there has been an increasing awareness of the value of tracheostomy in postoperative and post-traumatic care. During the past year there have been several excellent papers indicating the role of tracheostomy in a variety of situations where laryngeal obstruction is not the indication. Stead and Soucheray[3] suggested the use of tracheostomy for physiologic reasons in post-thoracoplasty care. Jensen[2] advocated the use of tracheostomy in the care of crushing injuries of the chest. Davis and Bishop[1] emphasized the role of tracheostomy in airway management in patients with poliomyelitis. It is the purpose of this paper to emphasize to physicians and surgeons the value of this simple procedure in the care of patients having a variety of conditions in which respiration is embarrassed due to obstruction of the lower air passages, impairment of respiratory physiology, and in prolonged unconscious states. The use of tracheostomy in surgery of the larynx and trachea are not within the scope of this paper.

Tracheostomy has usually been considered an emergency procedure to relieve acute laryngeal obstruction. It has a far greater usefulness as an elective procedure to give a ready avenue for aspiration of secretions in the lower tracheobronchial tree and to reduce the physiologic dead space in situations where respiration has been seriously impaired. It is well recognized that postoperative pulmonary complications are usually due to retention of secretions because of the inability of the patient to effectively expel them by the cough mechanism. Bronchoscopy and intratracheal aspiration with a catheter have been widely used in the clearing of retained secretions and prevention of postoperative complications. Both of these procedures, however, require a certain technical skill that cannot always be attained by nursing attendants. Aspiration through a tracheostomy tube, however, is a technic which is within the scope of any person of average intelligence. In situations where there is a continuing threat of the retention of secretions a tracheostomy is desirable.

PHYSIOLOGY

Ventilation of the lungs has two main purposes; (1) taking available oxygen to the pulmonary vascular bed for absorption into the blood stream and (2) to rid the body of carbon dioxide which is released by the blood into the alveolar air. In situations where respiratory function has been damaged either by paradoxical respiration secondary to a crushing injury to the chest or a too extensive thoracoplasty or where the musculature of respiration has been damaged as in

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Presented during the annual assembly of The Southwestern Surgical Congress, October 20-22, 1952, Dallas, Texas.

poliomyelitis, oxygenation of the blood may be augmented by the use of higher concentrations of inspired oxygen. This does not, however, remedy the inability of the body to rid itself of carbon dioxide due to decreased alveolar ventilation. The only way that alveolar ventilation can be improved is by reducing the physiologic dead space. This is best done as suggested by Stead and Soucheray[3] by tracheostomy.

INDICATIONS FOR TRACHEOSTOMY

Injuries to the trachea and bronchi. A sharp blow by a blunt instrument over the sternal notch or to the anterior chest may cause rupture of the trachea or the major bronchi without actually causing a penetrating wound of the chest. This injury is soon apparent due to the appearance of subcutaneous emphysema rising out of the mediastinum appearing first in the supraclavicular fossae. The patient develops a hacking, dry cough. With each cough the glottis is closed and air is forced out through the torn trachea or bronchi into the mediastinal tissues. As the process continues the subcutaneous emphysema may become life threatening. The same situation may arise with penetration of the trachea by small missiles or small stabbing instruments in which the skin closes rapidly. In these cases prompt tracheostomy is mandatory and will in most cases produce complete healing. An attempt to repair the torn trachea is usually not necessary, because as soon as a free airway is established, making it impossible for the patient to increase the intratracheal pressure, the tracheal injury will heal. If, however, there has been actual disruption of a bronchus a prompt thoracostomy should be done to repair the torn bronchus.

CRUSHING INJURIES OF THE CHEST

Paradoxical respiration due to a crushed chest or to too extensive thoracoplasty may impair breathing to a dangerous extent. To relieve this situation it is necessary to maintain an open airway and to employ measures designed to limit the paradoxical motion of the chest. Paradoxical respiration increases the physiologic dead space so that the patient is actually rebreathing alveolar air. If the difficulty of respiration is increasing notwithstanding the intratracheal aspiration and external splinting of the chest a tracheostomy should be done. This is especially desirable if there has been an accompanying head injury causing an unconscious state.

BRONCHOSCOPIC PROBLEMS

Tracheostomy may occasionally be required for subglottic edema following bronchoscopy for the removal of a foreign body in an infant or small child. Also, situations may arise during bronchoscopy in which it is necessary to do a tracheostomy at the time of the instrumentation. Foreign bodies are occasionally encountered in the bronchi which actually cannot pass through the glottic chink. A dry bean aspirated into the trachea or bronchus of a small child, after remaining in this moist air passage for a period of hours, will swell so that it may be impossible to draw it through the larynx. Actually, it is dangerous to attempt to

do so. The endoscopist in such a situation should do a tracheostomy with the bronchoscope in place. The bronchoscope should then be introduced through the tracheostomy opening and the large foreign body withdrawn through the tracheal incision. A tracheostomy tube should be put in place to remain there for at least two to three days. Broncholiths too large to be removed through the layrnx can be removed safely in the same manner.

UNCONSCIOUS STATES

Tracheostomy is very valuable in the care of patients who are in prolonged unconscious states. Neurosurgeons are frequently calling for tracheostomies in head injuries, intracranial operations, especially those affecting the eighth nerve, and cerebrovascular accidents. Patients unconscious from these conditions cannot effectively raise bronchial secretions and many of them die of pneumonia superimposed upon atelectasis. It is impractical to use bronchoscopy and repeated intratracheal aspiration through the nares. By doing a tracheostomy nurses in attendance can be trained to aspirate the lower trachea and bronchi to protect the lungs against infection.

PARALYTIC STATES

In paralytic states following injuries to the cervical and upper thoracic spine and in poliomyelitis, especially the bulbar type, tracheostomy should frequently be done. Many of these patients are not only unable to cough effectively but find it impossible to avoid aspirating pharyngeal secretions. Repeated aspirational episodes invariably lead to pulmonary suppuration, and in many cases to death. Tracheostomy provides an effective method of keeping the tracheobronchial tree clear and improves alveolar ventilation by reducing the physiologic dead space. It may tide these patients over a dangerous period so that if recovery occurs the lungs will not be damaged.

IN CHILDREN FOLLOWING OPERATIONS

It is impractical in the postoperative care of children who have had pulmonary resection for bronchiectasis or other suppurative pulmonary conditions to repeatedly use the bronchoscope or employ intratracheal aspirations. When bronchiectasis is bilateral or if there is any reason to suspect that retained secretions will be troublesome in the postoperative care, a tracheostomy should be done before the intratracheal tube which was used during anesthesia is removed. This insures an effective method of combating atelectasis and will greatly reduce the mortality and morbidity in performing such operations on infants and small children. It also spares the child repeated terrifying experiences.

TECHNIC

The technic of tracheostomy is simple. Certainly no surgeon and very few physicians should hesitate to do one when indicated. During the late war a medical corpsman received considerable notoriety and justifiable priase for doing a life

saving tracheostomy on a soldier who had received a penetrating wound of the neck on the battlefield.

A vertical incision is made from the cricoid cartilage to the notch of the sternum. By keeping the incision in the midline the ribbon muscles of the neck can be separated and the tracheal fascia exposed easily. Dissection of the tracheal fascia will expose the tracheal rings. The incision in the trachea should be made at least two or three rings below the cricoid cartilage. This is especially important in small children. In adults suffering from poliomyelitis, where care in a respirator may be necessary, it is permissible to make the incision through the trachea closer to the cricoid cartilage. In no case, however, should the cricoid cartilage itself be cut across. As soon as the tracheostomy opening has been made a tracheostomy tube should be selected which is approximately ½ the diameter of the trachea. A small wick of gauze should be placed down along the tracheostomy tube to allow drainage from the tissues of the neck. A tight closure around the tracheostomy tube is not desirable but two or three skin sutures are permissible if a long incision has been made. There is no objection to using a horizontal incision if one is careful in the dissection not to tear the dome of the pleura and thus cause a pneumothorax. As soon as the tracheostomy tube has been secured by tapes which encircle the neck, the tube should be aspirated carefully to remove all secretions and blood. One should routinely listen to both chests with a stethoscope to make sure that good breath sounds are present. If respiration remains difficult or there is any doubt as to whether the pleura has been torn a roentgenogram should be taken to determine whether or not a pneumothorax is present. If a pneumothorax is present it should be aspirated promptly.

TRACHEOSTOMY CARE

During the first few days following tracheostomy the dressing should be changed daily. The small wick which had been placed alongside the tracheostomy tube should be removed in 24 hours. The sutures in the skin, if any, should be removed in two or three days. The inner cannula should be removed every time the tracheostomy tube is aspirated and at such times it should be carefully cleaned, using applicator sticks or pipe cleaners. The cannula should not be left out of the tracheostomy tube except at the times of aspiration. The tracheostomy tube should be removed by the third postoperative day and cleaned. At this time there will usually be no difficulty in reinserting the tube into the trachea. Following this it should be removed at least twice a week for the first month. Occasionally tracheostomy tubes will have to be replaced by new ones when used in prolonged tracheostomy care if the original tube becomes unsightly due to tarnishing or if it disintegrates due to corroding.

DECANNALIZATION

Decannalization is one of the most important considerations in the care of a tracheostomized patient. In general, a tracheostomy tube can be removed as soon as the reason for instituting the tracheostomy no longer exists. Following

bronchoscopy tracheostomy tubes can be removed in two or three days. In injuries to the trachea and bronchi tubes should usually remain in place for at least a week. Care of injuries to the chest or in post-thoracoplasty care the tube should remain as long as paradoxical respiration is more than moderate. Obviously, in unconscious or paralytic states, the tracheostomy tube should be left in place until these states are no longer existent. Extreme care should be exercised in removing tracheostomy tubes in patients who have had bulbar poliomyelitis. This is especially true if there has been paralysis of the abductor laryngeal muscles. In many of these patients permanent tracheostomies will be necessary.

SUMMARY

The increased use of tracheostomy by physicians and surgeons in the care of patients having injuries to the trachea and bronchi, injuries to the chest, unconscious and paralytic states, and in children following thoracic surgery will prevent many pulmonary complications and prolong many lives. The technic and care of a tracheostomy is within the scope of every physician and his trained professional personnel.

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DEVELOPMENT OF TODAY'S CONCEPT IN THE TREATMENT OF HYPERTHYROIDISM

A REVIEW

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There are a great number of conflicting ideas according to the literature at present concerning the various treatments of hyperthyroidism. This has resulted in confused thinking by many physicians as well as by the laity. Our purpose is to present, not necessarily our own views on the correct treatment of hyperthyroidism, but to outline the consensus of opinions of others and to trace the development of the various types of treatment, as well as to outline their results.

For centuries, iodine in some form has been used in the treatment of goiter The ancient Chinese used burned seaweed poultices, while many South Americans chewed seaweed by the name of Palo-coto,[26] or goiter stick. Practica,[13] writting in 1170, advised the use of the ashes of sponge or seaweed. In 1820 Coindet[8] demonstrated that a goiter could be reduced in size by the use of the chemical iodine. In 1867 Luton[21] injected tincture of iodine directly into the gland. Following this experiment, the use of iodine fell into disrepute as noted by Theadore Kocher[20] who had seen unfavorable results. Following this renunciation of iodine by the old master Kocher, other and varied types of therapy were tried. The surgical technic was still so crude and was followed by poor results so frequently that considerable energy was expended in order to improve the mortality rate. By this time most technics included either general or local anesthesia as well as antiseptics. The effect of the work of Kocher, Mikulicz and Billroth had been acknowledged. Prior to 1870 many surgeons had been deterred from operating because of the fear of hemorrhage.

In 1906 boiling water was injected into the thyroid gland of patients with hyperthyroidism with the hope that the boiling water would destroy a part of the gland and result in a lowering of the secretion of thyroxin. This therapy produced areas of localized necrosis followed by scar tissue. In some patients it precipitated a thyroid crisis with its attendant high mortality. This practice was abandoned because it failed to adequately and safely lower the secretion of thyroxin so that the operation could be done with safety.

Roentgenotherapy was tried and, due to the low sensitivity of the thyroid tissue to roentgen rays, large doses were of necessity utilized. At that time x-ray machines were not so well calibrated nor were the doses well established. This led inevitably to occasional overdoses with serious reactions followed by ulceration and cicatrix formation. We recall 1 patient, a beautiful lady, who, because she did not want her neck scarred by surgery, took roentgenotherapy elsewhere which resulted in a horrible scarring of the skin. A stricture of the trachea developed and finally she died from an epithelioma which developed in the scar.

Today it is believed that roentgenotherapy from external sources is not the treatment of choice because of the low sensitivity of the thyroid tissue which necessitates large doses through the rather resistant skin of the neck. If such treatment were used with today's standardization and calibration of x-ray machines and technical knowledge, it should not be complicated by severe burns.

Reduction of the metabolism of the gland by reducing its blood supply, as is done in polar ligation, was at one time a very valuable prerequisite in many cases for partial thyroidectomy. Prior to the use of the antithyroid drugs polar ligation held a rather popular place as a prerequisite to thyroidectomy. There were some patients who developed uncontrolled crises following this minor procedure. A few years ago, regardless of the type of treatment, it was common to see a patient with thyroid crisis packed in ice in an attempt to control the high fever and rapid pulse which accompanied the crisis. Muttering delirium and uncontrollable restlessness of these patients presaged their early death.

One hundred years ago the mortality rate for thyroidectomy averaged about 45 per cent. Even 40 years ago the rate was high, varying from 6 to 17 per cent. In 1922 Henry Plummer[23] startled his contemporaries at the Mayo Clinic by advocating the use of Lugol's solution in the preparation of patients with hyperthyroidism for surgery. In 1924 following the use of Plummer's preoperative preparation, the reported death rate in a large series of cases was but 1.7 per cent.[23] With the use of Lugol's solution and the practice of doing thyroidectomy in stages, the mortality rate was lowered more even in the very severe cases.

In 1943 the next great step in the control of hyperthyroidism was taken when Astwood[3] introduced the goitrogen compounds. In hyperthyroidism there is an increased formation and secretion of thyroxin by the thyroid cells. The goitrogen drugs block this formation of thyroxin with a resultant lessening of secretion and lowering of the metabolic rate. In this process there is a compensatory hypertrophy of the acini and the gland due to the stimulus of the thyrotropic hormone. This results in further enlargement of the already enlarged gland. There is some variation in the reported rate of recurrence of hyperthyroidism in patients who are controlled by the goitrogens alone. Williams[29] reported that 51 per cent of his patients had a recurrence of hyperthyroidism within from two weeks to five months after the omission of the drug. McCullough[22] reported that only 35 per cent had recurrence in from 1 week through 14 months after omission of the drug. These recurrence rates, apparently, are the two extremes that are found in the literature. There are some patients who have reactions to these goitrogen drugs. The incidence of these reactions to thiouracil has been reported at about 10 per cent.[4] Cole[11] reported that 23.6 per cent of his patients had a reaction to thiouracil while only 3.85 per cent had a reaction to propylthiouracil. Cole stated that in his diffuse toxic goiters (he had a remission rate of 58.8 per cent while there was only 34.3 per cent remission in the nodular goiters) propylthiouracil, as we have just indicated, is much less toxic. Bartels[4] reported an incidence of 1.6 per cent of reactions. The incidence of agranulocytosis is variously reported from 0 to 1.74 per cent. Of these reactions we can find but two deaths reported in the literature. One was reported by Julian and Harris,[19]

which is not too well authenticated, and another was reported verbally to Dr. Astwood[2] by Dr. John McClintock of Albany, New York. Astwood[1] reported that 0.45 per cent of the first 5,745 patients taking thiouracil, whose records were reviewed by the Food and Drug Administration, died as a result of agranulocytosis. This occurred prior to the knowledge that this complication existed. The lives of some of these 23 patients might have been saved had the attending physicians been alert to this complication. The reaction rate of methyl-thiouracil, a drug which is little used in America, also shows a rate of about 10 per cent.[22]

Methyl-mercaptoimidizole (tapazole) is the newest and apparently the best[4] of the goitrogens. It is by far the most potent and the reaction rate is reported to be 4.8 per cent. Most of the reactions have involved the skin. There has been one incidence of agranulocytosis. We have used this drug extensively and have experienced but one mild skin reaction which was controlled by an antihistamine. While it is stronger than propylthiouracil, it does not seem to be any more active than adequate doses of propylthiouracil. In other words, the rapidity with which the disease may be controlled does not seem to be increased by its use. Since its mode of action is similar to the other goitrogens, it is assumed that the recurrence rate will be about the same.

Radioactive iodine was first prepared by Fermi[7] in 1934 and shortly thereafter its use as a physiologic aide, *i.e.*, tracer, was begun. In 1941 Hertz,[16] and Hamilton and Lawrence, reported its use in hyperthyroidism. The radioactive iodine (I-131) is concentrated in the thyroid gland and thus bombards the gland at close range with radiation from within. By this means, varying amounts of radiation may be applied directly to the cells of the gland. This radiation decreases the activity of the gland resulting in a decreased function with a lessened amount of thyroxin being formed and secreted. When using external x-radiation, the coverage is rather uniform but, in the use of radioactive iodine, some areas of the thyroid gland take up more of the radioactive iodine than other areas. This results in irregular deposition of I-131 in the thyroid and results in intense focal radiation in some areas while there is a lack of radiation in others. Of 475 patients treated with I-131, Soley[28] reported 83 per cent had good results, 9.7 per cent had fair results and 5.5 per cent failed to obtain the desired results. Seed and Jaffe[25] reported a recurrence rate of 3.5 per cent in diffuse goiter however, if the nodular goiters were included in the series, the recurrence rate rose to about 15 per cent. The *half life* of radioactive iodine is 8 days. This means that only half of the amount given originally is gone in 8 days and in another 8 days one half of the remaining radioactive iodine is gone. Thus, it takes many days longer than 8 for all of the radioactive iodine to be dissipated. It will require several years before it is possible to have a final evaluation of any harmful effects of internal beta radiation. It is generally thought that I-131 should not be given to young people. Crile[9] reported a case of amenorrhea following I-131 administration but did not make it clear whether this was due to direct action on the ovary or whether it was secondary to the hypothyroidism which developed. It has been postulated that, since I-131 is excreted through the kidneys, it may do damage to the renal function, but Haines[15] reported that there had been no

change in renal function in 40 cases which he thoroughly investigated. Freedburg[12] found no renal changes, as well as no hemopoietic changes, in patients taking I-131.

Haines[15] described a patient whose white blood cell count fell to 3,800 leukocytes per cu. mm., but returned to normal. He also described a patient whose basal metabolic rate rose from 46 to 62 per cent following the use of I-131 but he was not sure whether this was a result of radiation sickness or whether it was a mild hyperthyroid reaction, or both. Jaimet[13] reported that 6 women who had been given I-131 delivered normal babies. Gorbman[14] recorded the following undesirable effects in animals from very high doses of I-131:

1. Curtailment of growth.
2. Failure of function and regenerative properties of the thyroid.
3. Parathyroid injury.
4. Tracheal injury and tumors.
5. Recurrent nerve injury.
6. Ovarian sterilization.
7. Fatal adenohypophyseal tumors.

The relative dose to produce these results in mice was from as little as four times greater than the doses given to human beings to control hyperthyroidism, to as much as 20 times that amount. Freedburg[12] studied a group of patients who were given I-131 for heart failure. Some of these patients died and he obtained autopsies in 9. These autopsies were on the patients who had not died as a result of the I-131 but as the result of other disease and were obtained from 1 to 80 days after I-131 had been administered. These examinations showed no deleterious effects to any organ other than the thyroid. Sexton[27] reported that 1 patient died in thyroid crisis following administration of 11.5 millicuries of I-131.

It is thus believed that the use of I-131 should be limited to patients in the older age group, or those who, for some reason, are poor surgical risks. This should include patients with recurrent hyperthyroidism with unilateral recurrent pharyngeal nerve paralysis.

Surgery of the thyroid gland has gradually changed in recent years. The operation is no longer a bloody procedure, but one in which there is a strict observance of the anatomy involved with the ligation of arteries and veins before the gland is cut and the dissection and visualization of the recurrent laryngeal nerve. This procedure in conjunction with the preoperative preparation of patients with one of the goitrogens has reduced the mortality rate to an amazingly new low. Cole[11] has had no mortality since 1943. We have had none. Crotte[10] had a mortality rate of 0.3 per cent in his last 1,000 thyroidectomies. Cattell[6] reported that 0.25 per cent of his 1,630 patients from 1943 to 1949 died. He also stated that 1 per cent of his patients suffered recurrent laryngeal nerve paralysis, and 1.5 per cent had postoperative tetany. The recurrence rate was 2.4 per cent. There has been a gradual increase in the amount of gland tissue resected. Scott[24] reported that 17.5 per cent of his patients with diffuse toxic goiter, who had partial thyroidec-

tomy, had persistent manifestations of hyperthyroidism. These results led him to advocate total thyroidectomy. One fallacy of Scott's statistics may have been due to the lack of uniformity of the amount of thyroid gland removed. Cattell[5] has recognized that the recurrence rate is in general too high and at the 1952 meeting of the American Goiter Association reported that 4.7 per cent of 172 cases of recurrent goiter had a second recurrence after the second operation. Thus, he thinks that one must be rather radical in the amount of tissue which is removed. The policy of the Lahey Clinic still is to do a partial thyroidectomy but to do a very radical partial thyroidectomy, leaving less than 2 Gm. of thyroid tissue on each side. It is our belief that a near total thyroidectomy should be done and, since following this technic, our patients have had no recurrences.

SUMMARY

At present there are three fairly good methods for the control of hyperthyroidism. All three of these cause less morbidity and mortality than have any other forms of therapy for hyperthyroidism at any time in our history. Thus, all three must be considered good, however, there must be one which is better than the others.

The goitrogens are available almost anywhere and can be used by almost any physician who understands the physiology of the thyroid gland. Propylthiouracil has a reacting rate of 1.74 per cent. Tapazole has a higher rate but reactions are apparently mainly confined to the skin. The mortality rate is quite low. Several deaths have been reported but, if the patients are observed closely, the drug may be discontinued early enough to avoid mortality. The recurrence rate varies from 35 to 58.8 per cent in diffuse goiters.

I-131 is available only in centers owning expensive equipment and staffed by technically trained personnel. The reaction rate at the present time is unknown. One thyroid crisis following I-131 therapy has been reported. There may or may not be sterilization. There have been instances of severe hypothyroidism resulting from I-131 treatment but these were controlled with thyroid extract administration. The recurrence rate is about 3.5 per cent with 83 per cent obtaining good results and only about 5.5 per cent failures.

Operation is a satisfactory treatment of hyperthyroidism wherever surgeons trained in thyroid surgery are available. The only reaction to surgery in the well prepared patient is that following any operation. At the present time, thyroid crises are very rare. We occasionally hear of a case but such are not well authenticated. The mortality rate is about .1 of 1 per cent. Recurrence rate in partial thyroidectomy is about 2 per cent if a good radical partial thyroidectomy is done and, if a total thyroidectomy is done, there should be no recurrence.

We believe that the correct treatment for hyperthyroidism, whether it be in the diffuse or the nodular gland, should be preoperative preparation with the antithyroid drugs followed by operation at which time a total, or near total, thyroidectomy should be done. Radioactive iodine is an excellent means of control of hyperthyroidism in the older age group who are poor surgical risks.

We believe, at the present time, with the high rate of recurrence after the use of the goitrogens, that they should be utilized only as a preparation for surgery and not for the over-all treatment of the disease.

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FEMORAL EMBOLISM SIMULATING THROMBOPHLEBITIS

A CASE REPORT

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The diagnosis of peripheral arterial embolism is not always easy. Other conditions may closely simulate sudden embolic occlusion. The following case illustrates certain points which may aid in reaching a correct diagnosis.

CASE REPORT

M. W., a 36 year old Negro man truckdriver, was admitted to the John Gaston Hospital on Sept. 25, 1951, with the chief complaint of severe pain in the left thigh. Thirty six hours prior to admission he had felt a sudden sharp pain in the left upper thigh, and the left leg and thigh had rapidly become cold, numb and cyanotic. There was no past history of serious illness, and there was no history of rheumatic fever or of heart disease. In 1949 the patient had sustained an ice pick stab wound of the left chest in the fourth interspace near the posterior axillary line, but he had been discharged in good condition after nine days of hospitalization. A knife stab wound of the superior portion of the left buttock in 1950 had been treated in the emergency room of the John Gaston Hospital.

Two weeks prior to the current admission the patient had had malaise for about three days, and at the end of that time he had developed mild left lower chest pain which had lasted about six hours. Two days later, on arising, he expectorated a small quantity of sputum containing a *speck* of blood.

On examination the patient was in obvious pain. His temperature was 100.6 F. The heart sounds were normal and the chest was clear. There was diffuse cyanosis and coldness of the entire left lower extremity associated with moderate swelling and tenderness of the anterolateral aspect of the left thigh. Sensation in the extremity was diminished. The femoral pulse was greatly diminished and the popliteal, dorsalis pedis and posterior tibial pulse was absent. The left thigh was 3.5 inches larger in circumference than the right. The blood pressure was not obtainable in the left popliteal region but was recorded as 104/70 in the right.

The laboratory studies were essentially normal except for a white blood count of 22,400 per cu. mm. The serologic test for syphilis was negative. Roentgenologic examination of the left lower extremity was negative.

A tentative diagnosis of thrombophlebitis with arterial spasm was made, with embolism of the femoral artery or arterial spasm secondary to trauma being considered in the differential diagnosis. A paravertebral sympathetic block was done immediately after admission. Although there was some relief of pain, the response was considered to be poor. Another block eight hours later was much more successful, and there was immediate improvement in the color and temperature of the leg, although the pulse remained absent. Thereafter blocks were done as necessary to relieve pain and maintain warmth of the extremity.

Thirty six hours after admission the patient was noted to have developed slight swelling of the left calf associated with tenderness and a positive Homan's sign. The swelling in the thigh had diminished. Another paravertebral block done at that time was again successful. However, it was now believed that the admission diagnosis of phlegmasia cerulea dolens was probably correct, since it appeared unlikely that embolic disease would be so much improved by paravertebral blocks. Phlegmasia cerulea dolens represents a type of venous

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thrombosis occasionally seen in which severe arterial spasm is present and which may cause gangrene of the involved extremity.[1] This condition is also known as blue phlebitis, and it may simulate acute arterial occlusion. Pseudoembolic phlebitis is another term used to represent this particular type of thrombophlebitis. On the basis of this revised diagnosis the limb was elevated, and the next day swelling and tenderness in the calf were less pronounced. A barely perceptible left posterior tibial pulse was thought to be present. Nevertheless, in order to establish a positive diagnosis, venography and arteriography were done. A venogram on Oct. 4, 1951, showed no evidence of disease or obstruction in the veins of

FIG. 1.

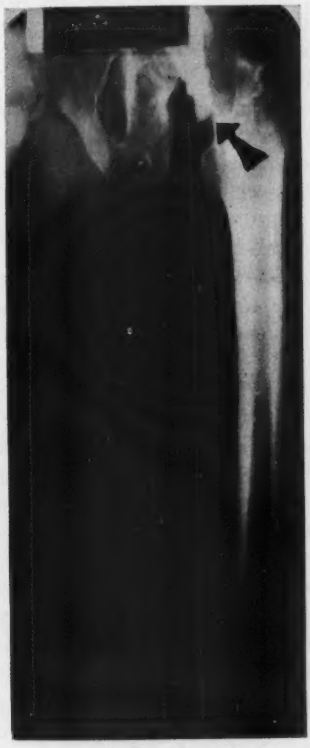

FIG. 2.

FIG. 1. Venogram showing patency of venous system

FIG. 2. Arteriogram showing complete occlusion of left femoral artery at the level of the bifurcation.

the left lower extremity (fig. 1). On October 9, an arteriogram showed a complete obstruction in the left femoral artery at its bifurcation (fig. 2).

As improvement progressed, the patient was permitted increasing activity about the ward. However, definite intermittent claudication was experienced after a very few steps. Accordingly, on Oct. 16, 1951, a left lumbar sympathectomy was done by Dr. James Bushart. Six days after operation, the blood pressure in each popliteal area was recorded as 134/90. The pulse was absent in the left dorsalis pedis artery but present in the popliteal and posterior tibial. The intermittent claudication was no longer present and the patient made an uneventful recovery.

DISCUSSION

Differential Diagnosis. The past history as well as the physical examination in this case failed to reveal heart disease, but, as has been pointed out by Pearse,[3] Wilson[4] and others, heart disease is not demonstrable in every case of arterial embolism. Pearse gave an incidence of 69.2 per cent in 296 cases where heart disease was demonstrated as the source of peripheral emboli. The most frequent types of cardiac disease are myocardial thrombi and mitral stenosis. The past history of a stab wound of the chest suggested that perhaps the origin of the embolus was an old pulmonary vein injury. The recent history of chest pain and questionable hemoptysis suggested pulmonary embolism from a phlebothrombosis or thrombophlebitis. The nature of the onset of the disease was compatible with the picture of blue phlebitis or arterial embolism. However, vasospasm associated with thrombophlebitis, although of frequent occurrence, is rarely severe enough to simulate arterial occlusion. De Takats[2] clearly stated in 1936 that acute vessel spasm accompanying thrombophlebitis may be mistaken for peripheral emboli.

Arteriography established the diagnosis of peripheral vascular embolism in our case. The saddle embolus obstructed both the deep and superficial branches of the femoral artery at the bifurcation. Pearse gave an incidence of 39.1 per cent of peripheral emboli lodging at this level.

Management: The conservative course which we employed was considered to be indicated because of the length of time which had elaspsed since the onset of the occlusion (36 hours) and the excellent response to sympathetic blocks, which rendered ischemic gangrene unlikely. The patient was placed on bed rest with the extremity kept slightly lower (3 inches) than the heart. The involved leg and foot were exposed to room temperature. Penicillin was given empirically. Anticoagulants were not used because of the possibility that retroperitoneal hemorrhage may result from the paravertebral blocks.

As the danger of gangrene passed and the patient was allowed to walk, it was discovered that calf pain was present on exercise. The collateral circulation, although adequate to maintain the extremity during rest, was inadequate during walking. Following sympathectomy, the collateral circulation was so improved that all pulse except the dorsalis pedis was present, and the blood pressure was equal in the popliteal areas.

SUMMARY

A case of femoral embolism simulating thrombophlebitis is reported. Certain difficulties in diagnosis and diagnostic procedures used are discussed. Angiography aided in establishing the diagnosis. Some problems encountered in treatment of this condition are briefly considered. Paravertebral blocks were effective and gangrene did not occur in a lower extremity in which the femoral artery had been occluded for 36 hours. Sympathectomy for the intermittent claudication following the acute phase of the disease produced a successful result.

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ACUTE INTESTINAL OBSTRUCTION

WITH SPECIAL REFERENCE TO ADHESIONS AND ADHESIVE BANDS

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The complexity of etiologic factors in small bowel obstruction makes its diagnosis and management a difficult and trying experience. Of leading significance in the patient with bowel obstruction is an abdominal scar.

A review of over 39,000 surgical cases from Massachusetts General Hospital, published by McIver[5] in 1932, disclosed 335 cases of bowel obstruction, of which 44 per cent were due to strangulated external hernias and approximately 24 per cent to postoperative adhesions and bands. In Cole's Textbook of Operative Technique,[2] Dennis of the University of Minnesota, states that 50 per cent of small bowel obstructions are due to external hernias and approximately 33 per cent are due to adhesions and adhesive bands. More recent studies, however, indicate that adhesions and adhesive bands are the predominant causes of intestinal obstruction today. In a recent series, published by Mersheimer and Winfield,[7] adhesions and bands were the principal causes of obstruction, accounting for 34 per cent of their cases. Extensive cancer surgery, with recent advances, particularly for malignancies within the abdominal cavity, is certain to raise the incidence of postoperative intestinal obstruction.

From a survey of patients with intestinal obstruction, admitted over a 10 year period at the University of Oklahoma Hospitals (1939–1949),[3] exclusive of strangulated hernias, 47 of 81 cases were due to adhesions and adhesive bands. However, careful scrutiny of the operative records in many of these cases failed to show the exact underlying cause of obstruction, and too frequently it was apparent that the operator also was not certain of the obstructing mechanism.

Fredrik Koch[4] has reported a series of cases of intestinal obstruction in which postoperative adhesions and adhesive bands were the causative factor in 66 per cent of the cases. He has classified adhesions and adhesive bands that lead to and produce obstruction of the bowel, into five etiologic groups:

1. Postoperative group which is the most common.
2. The congenital type.
3. Traumatic in origin.
4. Spontaneous or inflammatory in origin.
5. Multiple in origin, which is a combination of any of the former groups.

In Scandinavia, appendectomy was recorded as the most frequent predisposing cause of obstruction, and drainage of the abdominal cavity increased the incidence of ileus tenfold. Dennis states that adhesions and bands are approximately equal in occurrence and that more than half of the obstructions, due to bands

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Presented during the annual assembly of The Southwestern Surgical Congress, October 20–22, 1952, Dallas, Texas.

are sequelae of gynecologic surgery. In a recent report by Becker,[1] 90 per cent of a series of 412 cases of bowel obstruction due to adhesions had previously been subjected to laparotomy. Pelvic operations accounted for 34.3 per cent and appendectomy for 28.3 per cent of the cases.

A preponderance of adhesions and bands causing symptoms result from antecedent abdominal operations. Because of their related origin and frequent coexisting occurrence, adhesions and bands are usually grouped together as a cause of intestinal obstruction. When large areas of bowel are matted together the exact site of blockage and the exact mechanism of obstruction may be difficult to determine. However, despite their common origin and frequent coexistence, the morbid potential of the band, because of its ability to produce rapid strangulation, greatly over-shadows that of the adhesion.

With previous studies and attention being directed chiefly toward the physiologic changes produced by intestinal obstruction, the approach to more effective and direct therapy of the individual patient through accurate appraisal of the site and mechanical action by which the obstruction is produced, offers a favorable avenue of investigation toward which this discussion is directed.

BROAD BASE ADHESIONS

One of the natural responses of the peritoneum to insult, whether it be chemical, bacterial or traumatic, is the formation of a thickened cohesive surface which, by its adherence to adjacent surfaces, tends to wall-off or localize the insulting process. Such is the principle of healing of resected intestines. Adhesions assisting in the recovery of a minor insult are usually completely dissolved within a matter of a few weeks following resolution and healing at the local site. However, the greater the extent and degree of insult, and the more prolonged the process of healing, the more vigorous is the attempt of the peritoneum to localize the process and prevent widespread peritonitis by the formation of dense adhesions between adjacent structures. If healing at the local site is prolonged, the early fibrinous adhesions may be invaded or completely replaced by fibrous tissue, producing the *broad base adhesion* of postoperative origin. It may be situated between the antimesenteric border of a free loop of intestine and the denuded peritoneal surface produced by closure following intra-abdominal surgery. This type, *simple broad base adhesion*, may give rise to obstructive symptoms within a few days following laparotomy or after many years. Koch estimates that such adhesions persist after celiotomy in only 2 to 5 per cent of the cases.

Single mechanism of obstruction produced by broad base adhesions

With an element of kinking or angulation of the proximal intestine at the site of a *broad based adhesion*, simple mechanical intestinal obstruction, with its numerous and varying physiologic changes is likely to occur (figs. 1, 2). With impediment or blockage of downward passage of intestinal content, obstruction may occur high or low, and it may be complete or incomplete, acute or chronic. The function of the upper small bowel is secretory. The function of the lower

small bowel is principally absorptive. The first manifestation of a high simple obstruction is persistent vomiting producing marked dehydration and electrolyte imbalance. Because upper small bowel obstruction results in rapid regurgitation of the bowel contents, the characteristic distention seen in low small bowel obstruction is not present.

THE PHYSIOPATHOLOGY OF SIMPLE MECHANICAL LOW BOWEL OBSTRUCTION

The immediate effect of simple occlusion of the low small bowel is the emptying of the segment of bowel just below the level of the adhesive kink and distention of the segment of bowel immediately above the occlusion. The small bowel continues to propel its contents downward from above the site of block, producing an accumulation of gas and liquid, 68 per cent of which, Wangensteen[10] has shown to be swallowed air.

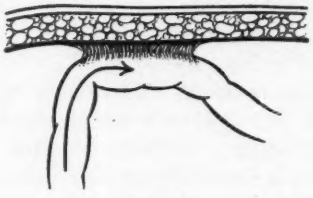

FIG. 1.

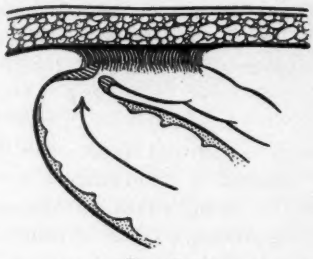

FIG. 2.

FIG. 1. Broad base adhesion between loop of small bowel and anterior abdominal wall at site of previous laparotomy.

FIG. 2. Diagrammatic sketch in cross section of broad adhesion with mechanical obstruction due to kinking. Local edema is exaggerated to show mechanism by which simple incomplete obstruction becomes complete. The valvulae conniventes are shown in the gas filled loop.

Dilatation of the segment of bowel immediately proximal to the site of obstruction serves as the afferent impulse for completion of a reflex for heightened intestinal activity. This reflex is mediated through the myenteric plexus and thus, the immediate result of the dilatation is an effective increase in the force of peristalsis.[9] This increased peristaltic force, in turn, actuates the painful intestinal cramps, commonly referred to as colic and ascribed by Wangensteen[10] to be one of the cardinal symptoms of intestinal obstruction. Associated with this hyperperistalis due to reflex stimulation, is an exaggeration of secretion within the bowel wall. Hence, the accumulation of swallowed air, the secretions of the proximal intestine, plus exaggerated local secretions of the glands in the bowel wall, produce a rise in intraluminal pressure. The increased intraluminal pressure slows capillary circulation and eventually leads to venous stasis and a degree of anoxia develops, resulting in alteration of osmotic pressure of the vessels and bowel wall. Fluid then pours into the lumen of the intestine and into tissues of the intestinal wall. Edema of the intestine rapidly supervenes to complete the obstruction.

As intraluminal pressure rises, so does dilatation increase, until reflex activity is lost, and the obstructed segment becomes atonic with only an occasional feeble peristaltic rush. At this stage, the high-pitched tinkling of the distended bowel is heard.

In this type of obstruction, if the first development, distention of the proximal loop, is not relieved, the distention will ascend, involving more proximal loops, until stagnation inevitably occurs. In this type of obstruction there is no immediate threat of gangrene of the bowel wall as in strangulating obstructions. Most authors agree that perforation occurs late in the disease and rarely causes death. A more subtle and insidious threat is the lethal toxins that may ultimately produce death. Unless open loop obstructions are decompressed, the sequence of impaired circulation, edema of the bowel wall and stagnation will cause enough damage to permit intraperitoneal spread of lethal toxins sufficiently to produce death.

TREATMENT OF OBSTRUCTION DUE TO BROAD BASE ADHESIONS

This type of obstruction is usually responsive to intestinal intubation. A weighted tube, introduced into the small bowel, will relieve the open loop type of distention and, with subsidence of edema in the intestinal wall at the obstructed site, the acute occlusion is usually relieved. A two to four day trial of conservative therapy is justifiable in the treatment of mechanical obstruction due to adhesions. Should the obstruction recur, relief by operative measures is advisable. Obstruction, occurring within the first three or four weeks following laparotomy, should be treated by intubation, inasmuch as the adhesions are pliable and stretching will relieve angulations of the bowel. McKittrick[6] has stated that all obstructions, seen within the first 24 hours, should be treated surgically. Many circumstances, however, make doubtful his attitude. If strangulation is not present, the *adhesion-former* and the patient with incurable abdominal malignancy are probably best treated conservatively.

ADHESIVE BANDS

Basically, the adhesive band, or inflammatory band is merely an attenuated broad adhesion, stretched into a fibrotic band from traction produced by the separation of adherent structures, originally held together by broad adhesion (figs. 3 and 4). In contrast to the *broad based adhesion*, the adhesive band, because of its strangulating capabilities, has a much greater lethal potential. Although the band may vary in size it is usually narrow and is always composed of dense avascular scar. It may serve as a connecting band between two loops of intestine; between a loop of intestine and the abdominal wall; between the mesentery and a loop of intestine; or between two leaves of the mesentery.

MECHANICAL ACTIONS OF ADHESIVE BANDS

In contrast to the broad base adhesion, which has only one obstructing action, the occluding capabilities of the adhesive band are more diverse and produce obstruction by any of four principal methods. The first two types of action are of extreme importance because of their potentialities toward early production of

circulatory impairment and strangulation of the bowel. The paramount danger in obstructions due to the adhesive band is the production of a *closed loop* obstruction.

The *closed loop* obstruction is produced by the herniation of a free loop of bowel through an abnormal hiatus composed of inelastic structures, formed in part, by the dense inelastic adhesive band. Because of interference with its blood supply, and particularly with venous return, and as a result of compression from the rigid walls, the herniating loop of bowel becomes swollen and incapable of reducing itself (fig. 5). The sequence of events in the closed loop type of intestinal obstruction is much more rapid than in simple obstructions due to adhesions. The action of the hiatus through which the bowel herniates is similar to that of the neck of the sac in a strangulating external hernia (fig. 6). Depending on the size of the rigid hiatus, the strangulating mechanism may be incomplete for an appreciable period of time in the *closed loop*, whereas, with a small un-

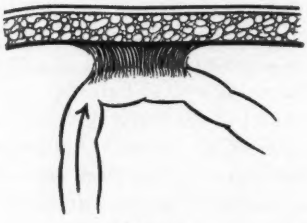

FIG. 3.

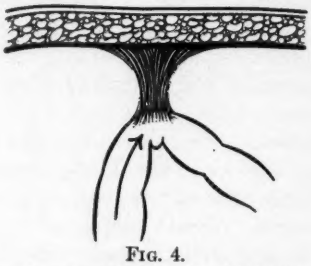

FIG. 4.

FIG. 3. Broad base adhesion which has begun to become attenuated due to pull of the bowel away from the laparotomy scar.

FIG. 4. Stretching and pulling of an adhesion to further fashion an adhesive band. Due to the abdominal distention, loops of bowel become adherent to areas that are normally separated in the undistended abdomen. This results in tension or stretching of the adhesions after the obstruction is relieved.

yielding hiatus, strangulation may be complete almost from the time of its inception and fulminating gangrene rapidly ensues.

The second mechanism of obstruction by the adhesive band which also produces early strangulation of the bowel is that due to volvulus. The *closed loop* in this particular type of obstruction results from torsion of the intestine upon its mesentery. The band serves to hold a loop to a rigid structure and permits the intestine to rotate and strangulate its mesentery. Bands exhibiting such action may be inflammatory, but are apt to be developmental anomalies, particularly related to a persistent omphalomesenteric duct.

The third obstructing mechanism of the adhesive band, rare in type, but occasionally seen, is induced by external pressure from an adhesive band passing close enough to occlude the lumen of the bowel by external compression. The obstruction produced, is again of the simple mechanical type and is usually incomplete or intermittent (fig. 7).

Similar to the singular action of the broad adhesion, the fourth obstructing

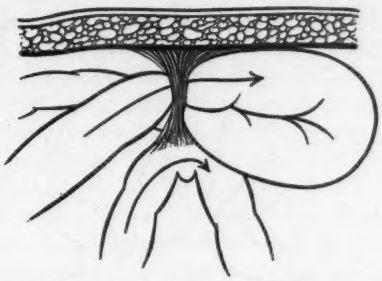

FIG. 5. Adhesive band and mechanism of action. Closed loop obstruction behind adhesive band. The hiatus through which the loop has herniated is formed by rigid structures. Circulatory impairment and gangrene may develop rapidly in the closed loop.

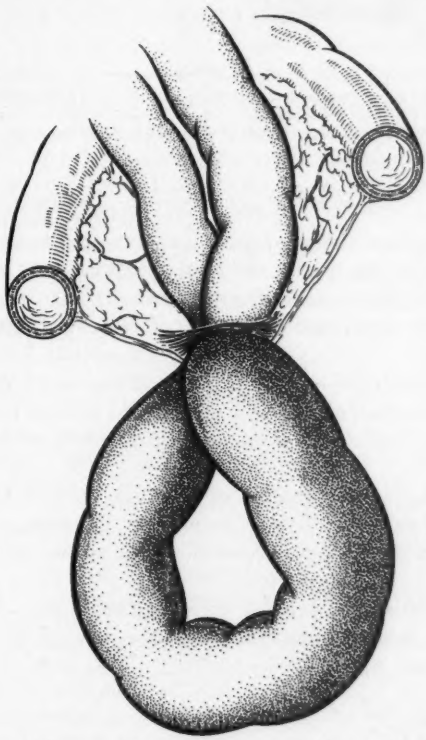

FIG. 6. S. F., 63 year old woman. Mechanism of closed loop obstruction with gangrene of ileum requiring resection. Hiatus through which the bowel has herniated is formed by an adhesive band and the leaves of mesentery of the small bowel.

mechanism is simply an angulation or kinking at its point of attachment to an unyielding organ, caused by an adverse pull on the bowel, from an extrinsic

source, the adhesive band. This type is not strangulating and can usually be relieved by intubation (fig. 7).

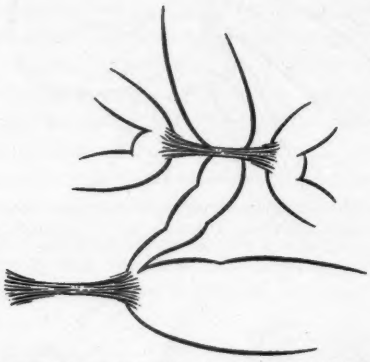

FIG. 7. P. A., age 63 years. Shows two adhesive bands with separate actions. Upper band producing obstruction by external compression and lower band by angulation and kinking. Mechanism of obstruction was visualized at operation four months after roentgenogram with barium.

SUMMARY OF OBSTRUCTING ACTIONS

The *broad base adhesion* produces only an open type of mechanical obstruction in which distention is progressive unless the angulation is relieved. Prolonged distention of the involved intestine may eventually lead to necrosis, but death is more often due to prolonged absorption of retained toxins from stagnation in the dilated loops.

Adhesive bands produce obstruction by any of four methods. The closed loop type of strangulation is the *bete noire* and should always be suspected and, if not quickly relieved, exploration is mandatory. The four mechanisms of obstruction are as follows:

1. The band aids in the formation of a hiatus, through which, a *closed loop* of bowel is trapped and immediately becomes strangulated.
2. Volvulus, a strangulating type produced by torsion of the intestine upon its mesentery.
3. Compression caused by extrinsic pressure from a band lying on the bowel.
4. Angulation or kinking from traction at the point of attachment to an unyielding organ.

DISCUSSION

From a practical viewpoint, the principle problem in the management of intestinal obstruction is the accurate and immediate differentiation between the strangulating and nonstrangulating types. In the strangulating type of obstruction, impaired circulation must be immediately restored if resection of gangrenous bowel is to be avoided. Wangensteen has shown that in a *complete closed loop strangulation*, gangrene and nonviability of the loop become complete

in three to four hours. Fortunately, development of complete strangulation in a closed loop is usually a gradual affair, allowing sufficient time for diagnosis before necrosis of the bowel wall occurs.

Accurate diagnosis of the *strangulating obstruction* requires careful repeated examination and accurate evaluation of symptoms. The onset of the disease is sudden and rapid. The pain is continuous and severe. The patient is *sick* from the inception of the disease. Vomiting is usually of an intractable nature and because the *closed loop* does not permit the passage of sanguinous content, the vomitus does not contain either old or new blood. Abdominal tenderness is usually quite marked and rebound tenderness is a valuable diagnostic sign in strangulating obstructions. Bowel sounds are diminished or absent early in the disease, particularly if loss of viability occurs rapidly. Elevation of the pulse and temperature are especially confirmatory in determining loss of viability of the bowel in the more slowly progressive types of necrosis.

Pathognomonic of strangulation is the presence of a palpable mass which sometimes is not detectable until the abdomen is relaxed by anesthesia. Due to accumulated fluid, the gangrenous loops are frequently deep in the abdominal cavity. The roentgenogram is a valuable asset in determining the site of the obstruction and detecting early signs of obstruction producing gangrene. Important as it may be, however, it cannot suffice for repeated bedside examination, but must be correlated with the clinical picture.

Rigler[8] lists as helpful signs in the diagnosing of strangulating obstructions:

1. The presence of a gas filled, coffee-bean type, *closed loop*.
2. The fluid filled loop, producing a soft tissue shadow (pseudotumor).
3. Failure of gas filled loops to rise in the erect roentgenogram.
4. Loss of mucosal pattern in a distended loop.

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THE RECTAL TUBE AS A LETHAL INSTRUMENT

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Injury to and through the rectum may occur in five general ways:

1. As a gunshot wound
2. From an impalement
3. By compressed air or water
4. As a submucosal enema
5. From the perforation by an enema or diagnostic instrument into free abdominal cavity.

We wish to review briefly this subject and report 3 new cases of perforation of the bowel with enema tubes.

During combat, explosives and shell fragments may perforate the lower bowel as a part of a complicated injury wherein abdominal wall, mesentery and adjacent organs are involved. Shock is often severe, and foreign bodies are frequently present. Under field conditions, repair of the injured bowel, control of hemorrhage, and removal of the detritus must often be accompanied by a proximally divided colostomy. Drainage of the presacral space is necessary. The mortality rate in these patients in World War II was approximately 25 per cent.[2, 10]

Severe impalement injuries in civil life imitate but do not duplicate those of war. The causal agent is single and blunt. It is usually a stick or a diagnostic instrument. Many of these cases occur in children when they fall or slide onto sticks or rake handles. Though a single organ usually is involved, diagnosis may be difficult. The same principles of treatment are the same as in gunshot wounds. Impalement victims almost all recover.[12]

Pranksters using pneumatic equipment may blow out their fellow's bowels in attempting mawkish jokes. When a stream of compressed air is directed at the anus it sometimes enters the sphincter, and passing under high pressure to the unencased rectosigmoid, splits it open in many places. The fecal contents are spewed throughout the peritoneal cavity. Notwithstanding swift repair, with a proximal colostomy if indicated, the mortality rate is excessive.[9]

Although it is not strictly closely allied to this discussion, we cannot omit a report of Adolph's 60 year old man who had developed a peculiar method of moving his bowels. He was accustomed to inserting an ordinary garden hose into his rectum and turning on the water tap until the desired pressure was felt. One day his hand slipped and he flooded his entire peritoneal cavity with water and feces through a 3 cm. tear in the sigmoid. He recovered after closure of the bowel and drainage of the abdomen.[11]

The submucosal administration of enema solutions is apparently more common in England than in America. There are reports in the British literature of enema tube tips penetrating between the mucous membrane of the rectum and the

underlying tissues. The fluid injected dissected up and around the rectum and urogenital outlet, carrying with it the dissolved salts and soda. Indolent sloughs resulted with prolonged disability. Many patients lost all their sphincter control and some were grieviously ill. For example: 1 case is reported of a normal postpartum woman who was so injured. Her entire rectovaginal area became necrotic and was ultimately discharged as grumous waste. After a long convalescence with a defunctioning, permanent colostomy, recovery occurred. The reported high mortality in the past in such cases would undoubtedly be less with modern antibiotics and supportive therapy.[7, 8]

It is hard to understand how such a traumatic indignity could be perpetrated upon a conscious patient by a responsible attendant. Nevertheless, it frequently has been done with tragic results for everyone concerned including patient, nurse, physician and hospital. We must train our nurses and physicians to avoid such mishaps, and we must also train them to recognize such accidents if they do occur. An occasional conscientious physician has reported his experience when his sigmoidoscope perforated the bowel. One such report tells of viewing the gallbaldder per rectum.

Perforation into the free peritoneal cavity with an enema nozzle is a rare surgical emergency which can if recognized early, be treated with a low mortality. We review 7 cases from the literature and add 3 from our personal experience. Our first 2 cases, interestingly enough, were seen within 2 weeks of each other.

When the enema tip penetrates the intact bowel, it usually enters through the wall of the rectosigmoid. Perforation in this location is common because the curve of the bowel directs the enema tip. The involved area is insensitive, and a perforation may be made in normal people without excessive force. The administration of the fluid into the peritoneal cavity causes immediate severe pain localized in the lower abdomen on the left side. Fecal contents are carried along to grossly contaminate this area. Mild shock may ensue, yet some people do not call for medical aid until suppuration develops and pain becomes intense. Physical findings vary, depending upon the time after the accident, but the classical signs of infection and peritoneal irritation are to be expected. Abdominal tenderness and rigidity are common. Air beneath the diaphragm may be found by roentgenogram. Immediate exploration is necessary. At operation the perforation can be seen as a *powder burn* spot on the antimesenteric side of the bowel. Repair is easy. Simple closure is effective. Following early operation the abdomen may be safely closed without drainage. Antibiotics should be given postoperatively. If surgery has been unduly delayed, drainage may be necessary and secondary infection is more likely to follow.

Case Reports

Our first patient, Mr. C. W. M., a 61 year old farmer had had an abdominoperineal resection for a rectal carcinoma one year before. Thirty-six hours before admission on Dec. 25, 1951, he attempted to give himself a cleansing enema through the colostomy. When the soft rubber catheter was inserted into the bowel, it encountered resistance. He pushed hard and felt it pass through a yielding wall. He immediately had moderate pain in the left

lower abdomen, but this quickly subsided. He irrigated the catheter without results and the pain recurred. In 12 hours he had generalized abdominal cramping, with pain localized in the right lower quadrant of the abdomen. He was referred to us 36 hours following the accident with a tentative diagnosis of either appendicitis or perforation of the bowel by a catheter tip. When he entered the hospital he had a temperature of 103 F., respiration of 20 and a pulse rate of 80. There was exquisite tenderness in the right lower abdomen and some in the left abdomen. The abdomen was not distended and peristalsis was heard. The pulse was irregular, and the blood pressure was 145/98. There were no other pertinent physical findings. Urinalysis, red cell count and hemoglobin were normal. The white cell count was 8,000 leukocytes per cu. mm. with 66 per cent polymorphonuclears. At operation the appendix was found to be normal with some periappendicitis, presumably due to the enema solution. A *powder burn* appearance on the antimesenteric border of the sigmoid, 3 inches proximal to the colostomy, marked the site of perforation. There was very little peritoneal reaction. No cancer was seen or felt anywhere. The perforation was easily closed with interrupted cotton sutures and the abdomen was closed without drainage. After an uneventful postoperative course, he was discharged on the eighth postoperative day and has been well since.

Our second patient, a dentist, was a man age 71. At 4 p.m. on Jan. 11, 1952, he gave himself an enema through a hard rubber catheter. He experienced immediate pain and discharged bright red blood from the rectum. He was examined two hours later. The physical examination was negative except for diffuse abdominal tenderness, somewhat localized in the left lower quadrant of the abdomen. The urinalysis, red cell count and hemoglobin were normal. Roentgenogram showed free air beneath the diaphragm. The white cell count was 4,950 leukocytes per cu. mm. with 69 per cent polymorphonuclears. At operation, a typical *powder burn* perforation of the anterior surface of the rectosigmoid was found and closed. The abdomen was closed without drainage and after an uneventful convalescence he was discharged on the ninth postoperative day.

Our third patient, Mrs. I. D. B. was a 70 year old white American housewife. She had an abdominoperineal resection elsewhere on Jan. 25, 1951, for carcinoma of the rectum. The colostomy closed and she became constipated. About six hours before admission to the hospital on Dec. 27, 1952 she gave herself an enema. Following this she had severe abdominal pain and was at once admitted to the hospital with mild distention. Routine laboratory data was within normal limits, as were her vital signs.

Physical examination revealed diffuse abdominal tenderness; and a preoperative diagnosis of bowel perforation was made.

At operation through a left rectus incision she was found to have perforated the terminal colon on the antimesenteric border about 3 inches from the outlet. There was a great deal of fecal material contaminating the abdomen with a typical powder burn appearance at the site of injury. The splenic flexure was mobilized and a new colostomy fashioned for her. After a somewhat stormy postoperative course she was discharged on the twelfth postoperative day.

Summary of Reported Cases

These case reports here summarized are all the well documented cases of perforation into the free abdomen by enema tips which we have found.

Case 1. Nugent, in 1833, reported a boy infant of 1 year of age who was given an enema with intraperitoneal injection of the solution. The child soon died of peritonitis.[4]

Case 2. Behrend and Herman, 100 years later, operated upon a patient who seven hours before had received an intraperitoneal enema. A perforation of the sigmoid 28 cm. from the

anal orifice was found and repaired. This patient died four hours postoperatively of peritonitis and toxemia.[1]

Cases 3 and 4. Pearse in reporting "8 cases of perforation of the bowel by a foreign body inserted through the anus" mentions 2 caused by enema tips. One, a 71 year old man, perforated the sigmoid by giving himself an enema. Notwithstanding supportive measures, operative closure of the rent and proximal colostomy, he died. The second, a 69 year old man who did the same thing, survived. In this case, closure of the perforation was done within three hours after the accident and no colostomy was deemed necessary.[5]

Cases 5 and 6. Pratt and Jackman reported a 63 year old man who gave himself an enema. On evacuation he had severe pain and abdominal tenderness. At operation a typical sigmoid perforation was found and closed. He made an uneventful recovery. These same authors had a 66 year old woman who received a high colonic irrigation three months before they saw her. Her course had been very stormy with many abscesses and two exploratory operations. She developed a stricture which had to be dilated. She made a spontaneous recovery.[1]

Case 7. Gabriel[3] reported a 57 year old man who perforated his sigmoid colon with an enema tip. Notwithstanding exploration within 12 hours, he died.

COMMENT

The history, physical findings and pathology in all our cases of perforation by an enema tip through the bowel into the free abdominal cavity followed a characteristic pattern. The patient was usually a normal adult, often well skilled in self administration of enemas.

The perforation is made without undue force and is followed by severe but not incapacitating pain. These patients have tenderness, muscle spasm, and sometimes rigidity in the lower portion of the abdomen, with an elevation of the white cell count. Roentgenograms demonstrate air beneath the diaphragm. At operation a *powder burn* appearance of the rent in the bowel is seen in the free sigmoid at its antimesenteric border. Immediate closure of the perforation using modern aids warrants a good prognosis. Any surgeon will see few such cases, but it is well to remember they can occur. Nurses, patients and professional or amateur bowel cleansers should be warned of the danger of bowel perforation.

SUMMARY

We present a brief discussion of the origin and treatment of some common injuries to and through the rectum. It is noted that perforation of the rectum by an enema tip is easy and that it occurs in normal people. With prompt treatment patients usually will recover.

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BLEEDING GASTRIC ULCER REQUIRING PARTIAL GASTRECTOMY IN A PATIENT RECEIVING CORTISONE

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The effect of ACTH and cortisone on the body tissues, both normal and diseased, is still in the investigative stage. There is indication that these drugs may produce, or at least reactivate, certain disease processes. Likewise the healing power of tissues in patients receiving these drugs may be retarded. With these thoughts in mind, what attitude should the surgeon take when faced with the necessity of performing major operations upon patients receiving treatment with these drugs? We have recently had under our care a patient receiving cortisone for exfoliative dermatitis and psoriasis who developed severe upper gastrointestinal bleeding, necessitating emergency partial gastric resection. Our experience in the care of this patient might be of interest to those faced with the prospect of operating upon patients being treated with ACTH or cortisone.

Case Report

L. I., a 61 year old white man gardener, was admitted to the hospital on Aug. 11, 1951 for treatment of a severe exfoliative dermatitis, complicated by pustular psoriasis, and thought to be due to lead and arsenic poisoning. There was a history of operation for a perforated duodenal ulcer in March 1950, with a wound disruption on the ninth postoperative day. Since that time he has had no stomach complaints requiring treatment. During the next eight months he was treated both systemically and locally in the dermatology service. Shortly after admission ACTH in doses of 20 milligrams four times per day was started and continued. There was some improvement after six months of treatment. On March 6, 1952 the ACTH was discontinued and cortisone, 150 milligrams every 12 hours, was begun. Definite improvement followed. On April 14, 1952 the cortisone dose was reduced to 100 milligrams twice daily. Attempts to reduce the dose further resulted in an exacerbation of the exfoliative dermatitis. On this regime of cortisone combined with local therapy and general supportive measures, he showed some improvement but was not considered sufficiently improved to warrant discharge from the hospital.

On May 3, 1952 the patient complained of dyspnea and dizziness and fell in the hallway. Questioning revealed that he had noted a burning sensation in the epigastrium for the previous two or three days and had had one black stool. The hemoglobin was found to be 4.5 Gm. per 100 cc., red blood cell count 1,390,000 per cu. mm. Cortisone was discontinued and 1,000 cc. of whole blood were given. On the following day the hemoglobin was 6.8 Gm. per 100 cc. and another 1,000 cc. of whole blood were given. On May 6, 1952 he vomited

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blood, complained of epigastric gnawing relieved by food and had a stool consisting chiefly of clotted blood. The hemoglobin had dropped 5.15 Gm. per 100 cc. He was given 2,000 cc. of whole blood with vitamin K in large doses in preparation for surgery. The blood chemistry studies made prior to operation revealed a blood urea nitrogen of 56 mg. per cent, serum protein 3.75 Gm. per cent, plasma chlorides 103 mEq. per liter, CO_2 27.9 mEq. per liter, and total base 162 mEq. per liter. On the fourth day of his acute illness partial gastric resection was done. No definite bleeding site could be found and there was no blood in the duodenum or the jejunum. The duodenum was thickened and scarred but did not appear acutely inflamed. Some difficulty was encountered in freeing the lesser curvature of the stomach although the stomach in this region did not appear chronically or acutely inflamed. Examination of the removed distal 3/5 of the stomach revealed an acute gastric ulcer approximately 3 cm. in diameter on the lesser curvature. The ulcer crater was shallow and flattened with very little surrounding induration. A retrocolic Polya anastomosis was made. Fifteen hundred cc. of blood were given during the operation. With supportive treatment, including cortisone 50 milligrams twice daily, convalescence was remarkably uneventful until the evening of the tenth postoperative day when the patient complained of sudden severe pain in the midabdomen. There was generalized abdominal tenderness more severe in the lower abdomen and moderate muscle spasm. A diagnosis of leakage from the duodenal stump or from the anastomosis line was made. At abdominal exploration approximately 250 cc. of serosanguineous fluid was aspirated from the peritoneal cavity. The site of the complication was neither the duodenal stump nor the anastomosis line. Apparently a small collection of seropurulent exudate had developed in the lower portion of the incision and ruptured into the peritoneal cavity. A small drain was placed in the area. Further convalescence was uneventful. The patient is now doing well on a low sodium, high protein diet with supplementary potassium chloride. He has continued to receive 50 milligrams of cortisone twice daily with increased improvement of the exfoliative dermatitis. When last seen six months following operation he had no difficulties referable to the gastrointestinal tract, but continued to be a problem in dermatological management.

DISCUSSION

Two main points of discussion are raised by this case report: first, the possible role of cortisone or ACTH in the production or activation of peptic ulcer, and second, the effect of cortisone on wound healing and the ability of patients to withstand major surgical procedures.

Concerning the first point, there is considerable difference of opinion. In a study conducted by Mann[9] of the side effects on 4,376 patients using ACTH, 74 or 1.7 per cent developed perforation. These figures certainly seem to be significant both as to the percentage of patients developing ulcer and the incidence of perforations, although figures on a similar control group are not available. Numerous case reports of peptic ulcer, both gastric and duodenal, appearing or being activated during treatment with both ACTH and cortisone have been recorded.[2, 5, 7, 8, 10, 13, 14] Gray et al[4, 15] found that ACTH administration produced an increase in gastric pepsin as well as an increased uropepsin excretion and suggest that the gastric acidity is increased. Similar findings are reported by Kirsner.[7] Thorn,[1] on the other hand, states that the gastric acidity remains unchanged after giving ACTH to normal subjects. Sandweiss[13] found a higher nocturnal secretion of free hydrochloric acid after cortisone treatment than before.

In view of the above reports it would seem advisable to proceed with caution when giving ACTH or cortisone to patients with a known ulcer diathesis. The

concurrent usage of antacids and antispasmodic drugs might neutralize the bad effects, if any, of these drugs.

The effect of ACTH and cortisone on wound healing seems to depend on the dose and the nutritional state of the patient. Ragan, et al[6, 11, 12] have conducted extensive studies on rabbits and have demonstrated a delay in the appearance of granulations as well as a delay in wound healing when rather large doses of ACTH or cortisone were given. They also have observed a similar delay in wound healing in human patients and reported diminished reaction in the presence of infection. Similar results were obtained by Taubenhaus[16] who found that cortisone and ACTH may cause a delay in development of all elements of connective tissue. Thorn[18] has also pointed out the inhibitory effect of adrenocortical steroids on wound healing and fibroblastic proliferation. However, recently published studies by Baldridge et al[1] on in vitro effects of cortisone on mesodermal tissues showed that proliferation of embryonic fibroblasts in the chick embryo is not inhibited by the drug. They also found no direct in vitro effect on the viability of circulating human leukocytes. Likewise, recent studies made by Taylor et al[17], using rats, and by Cole et al[3], using dogs, have failed to demonstrate any constant retardation of wound healing or fibroplasia in those animals receiving cortisone in doses comparable to therapeutic doses in man. The last group stated that they have operated upon patients receiving cortisone treatment and observed no delay in wound healing.

Present indications are that the usual dose of ACTH or cortisone causes little interference in the healing of primary sutured wounds. There likewise seems to be little experimental or clinical evidence to support statements that intestinal anastomoses are more apt to disrupt in patients receiving these drugs in the usual therapeutic doses. Careful attention to the patient's nutritional and electrolyte status with gentle and precise surgical technic seems of far greater importance to wound healing than the effects of ACTH or cortisone when used in therapeutic doses. Retention of water, sodium and chloride with an increased excretion of potassium in patients receiving ACTH and to a lesser degree in those receiving cortisone makes necessary close attention to fluid and electrolyte balance in the postoperative period. Also, since these patients are usually in negative nitrogen balance, an increased intake of protein is indicated.

Although conclusions should not be drawn from 1 case, experience with the patient here described indicates that operations may be done on patients being treated with cortisone without fear of their ability to either withstand surgery or to heal properly. In the patient here recorded, continuation of cortisone in moderate doses during the postoperative period seemed to have no undesirable effects.

SUMMARY

A case of bleeding gastric ulcer necessitating partial gastrectomy in a patient on prolonged cortisone therapy is presented with comments and discussion of the problems confronted.

Although the possible role of cortisone and ACTH in the production or re-

activation of peptic ulcer is not yet clear, it seems advisable to proceed with caution when giving these drugs to patients with a known ulcer history.

When patients receiving these drugs are operated upon, careful attention to nutritional and electrolyte needs, as well as gentle and precise surgical technics, seems of greater importance to wound healing and early recovery than the influence of cortisone and ACTH when used in the usual therapeutic doses.

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APPENDICEAL CALCULI†

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The mortality rate in appendicitis has decreased markedly and steadily from 1930 to the present date. The deaths in the United States of America due to appendicitis have decreased as indicated below:

1933	17,717‡
1935	16,142
1940	12,999
1945	6,699
1949	3,744
1950	3,110

Obviously the cause for the progressive decline in mortality for appendicitis is due to many factors, some of which are the improved antibiotics, better understanding of fluid and electrolyte balance, public health campaigns, properly selected anesthetics, adequate blood for transfusion, and more widespread availability of surgeons with formal training. Further improvement in the mortality rate will depend chiefly on more information on fundamental surgical problems but also to some extent on the accurate diagnosis of appendicitis when it simulates a disease that does not necessarily demand prompt surgical exploration. This paper is written to give emphasis to the occurrence of appendiceal calculi, for such calculi may be erroneously interpreted as being of other than appendiceal origin. Some of the calcified bodies with which appendiceal calculi may be confused are: 1. Ureteral calculi. 2. Renal stones. 3. Vesical stones. 4. Gallstones in the gallbladder or impacted in the lower ileum. 5. Calcified mesenteric lymph nodes. 6. Phleboliths. 7. Retained barium. 8. Calcified dermoids of the ovary. 9. Calcified appendices epiploicae. 10. Enteroliths. 11. Appendiceal foreign bodies.

Appendiceal calcluli differ from appendiceal concretions or fecaliths. Fecaliths are nonopaque in roentgenograms. Fecaliths statistically have an incidence[15] of from 5 to 80 per cent, depending upon the type of disturbance found in the appendix.

Since the original description of appendicitis by Reginald Fitz, in 1886, observers have repeatedly confirmed his finding of the incidence, of fecal concretions in the appendix, especially in the cases with gangrene and perforation to be 59 per cent in the autopsies performed by him.[2] Wangensteen and Bowers, in a recent study of acute appendicitis, found fecaliths in 44 per cent of the suppurative cases, and in 80 per cent of the cases with gangrene[2].

On the other hand, appendiceal calculi are radiopaque concretions (as demon-

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‡ These statistics were secured from the National Office of Vital Statistics and the Metropolitan Life Insurance Company.

strated by a plain roentgenogram of the abdomen) and are considered by many to be rare. Bunch and Adcock found only one true appendiceal calculus in a series of over 2,000 cases studied. According to Felson and Bernard in 1947, slightly over 100 cases have been reported since 1900, the majority of which were in the foreign literature. We consider the investigations carried out by Felson in 1949, and the high percentages (12 per cent) relative to the incidence of appendiceal calculi as being open to question.[4, 8] Felson made roentgenograms of the appendices after removal and calculated whether the radiopaque material would be visible in a plain roentgenogram of the abdomen. This introduces an uncontrollable number of variables.

Weisflog in 1906, was probably the first person to diagnose appendiceal lithiasis preoperatively.[22] Most frequently, the presence of the calcified mass in the appendix found at operation was not suspected. Very few authors have made a correct preoperative diagnosis[4]. The various confusing calcified bodies have been listed above. Appendiceal calculi originate in the appendix. Occasionally, the nucleus of the stone is a foreign body. Most usually, however, the center of the stone is mainly vegetable pulp containing mucus and large numbers of bacteria, but no cholesterol, bile-salts, or pigments. The hard outer shell, which not infrequently is laminated, is composed chiefly of calcium phosphate and some magnesium phosphate.[4] Their chemical composition, accordingly, differs from that of gallstones and true enteroliths.

Appendiceal calculi may be single or multiple and faceted, solid or laminated. Generally they are circular and smooth as opposed to the spotty calcification frequently seen in calcified mesenteric lymph nodes. They are usually larger than the lumen of the appendix and vary in size from 0.4 cm. to 4.0 cm. in their longest diameter.[16] Stones weighing as much as 13.5 Gm^2 have been reported. The degree of mobility of a stone within the lumen of the appendix or mobility of the appendix itself may aid in the differential diagnosis. Fixation of the appendix is usual, due to the associated inflammation. A lateral roentgenogram of the abdomen will frequently show these stones to be located too far anteriorly for ureteral calculi.

A calculus in the appendix, opaque in a plain roentgenogram, is of sufficient rare occurrence to make a study of cases helpful in the avoidance of diagnostic error.[5] In the three and one-half years from July 1, 1948, to Dec. 31, 1951, we have operated upon 3 cases of appendiceal calculi in a total of 185 appendectomies. The first case was not diagnosed correctly clinically but was diagnosed by the roentgenologist. A calcified mesenteric lymph node and a foreign body were considered. The second and third cases were diagnosed preoperatively by the clinicians upon seeing the roentgenograms. Our percentage occurrence of proved appendiceal calculi, as seen by a plain roentgenogram of the abdomen before appendectomy in this series is 1.6 per cent. This series is probably too small for statistical significance, but has led us to the opinion that calcific disease of the appendix is not as rare as is commonly thought; but also probably is not so common as some series may indicate.[8] With the condition constantly in mind, it is not a difficult preoperative diagnosis to make. We believe that if plain roent-

genograms were a more frequent part of the diagnostic investigation in patients suspected of having appendictis (and certainly such roentgenograms most usually are not necessary), the entity of appendiceal calcluli would be seen more frequently. Of our 185 cases of appendectomy, 89 or 47.8 per cent, had plain roentgenograms of the abdomen. There were 55 cases of appendicitis that were sup-

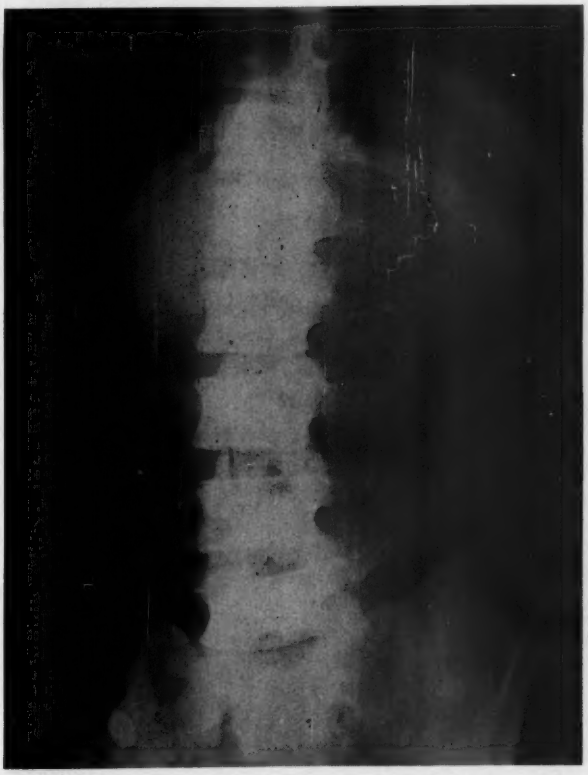

FIG. 1. Case 1. Roentgenogram taken before operation showing a solitary appendiceal calculus.

purative or perforated, and 32, or 58 per cent, of these had plain roentgenograms of the abdomen. The distribution of the pathologic types of appendicitis in our series was as follows:

Acute appendicitis	62
Acute phase of recurrent appendicitis	52
Acute suppurative appendicitis	19
Gangrenous appendicitis	13
Perforated appendix with abscess	22
Perforated appendix with spreading peritonitis	10
Nonpathologic	6

There were two deaths in this group. One patient was not operated upon. All of the patients with appendiceal calculi survived, though their pathologic type of appendicitis was severe.

Case 1. E. C., a Negro male, age 42 years, was admitted to the hospital on April 5, 1949, with the complaint of a mass in the right lower quadrant of the abdomen, which was noticed

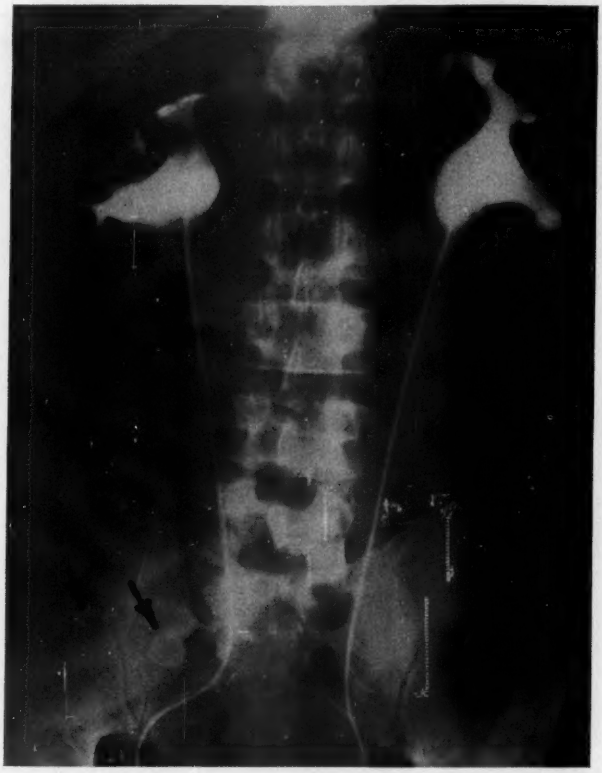

FIG. 2. Case 2. Retrograde pyelogram showing the two appendiceal calculi obviously outside the urinary system. On a subsequent roentgenogram (not shown here) the two stones were closer together, the smaller one was mobile within the lumen of the appendix.

one month previously by his wife during a massage. He had had some infra-umbilical pain one month prior to admission but was presently free of discomfort. There was a firm mass 3 cm. in diameter palpable just above the lateral third of Poupart's ligament. Clinically, the mass was thought to be a calcified lymph node or foreign body, but the roentgenologist correctly diagnosed it as an appendiceal stone and mentioned the possibility of a foreign body. The admission urinalysis was normal. The hemoglobin was 16.0 Gm. per 100 cc. and the leukocyte count was 6,600 per cu. mm. Stool examinations were negative for blood. Laparotomy, using a McBurney incision, revealed a perforated appendix containing an appendiceal calculus 1.8 cm. by 0.8 cm. well walled off. An appendectomy was done and the patient had an uneventful convalescence (fig. 1).

Case 2. J. W., a negro male, age 36 years was admitted to the hospital July 11, 1950 on the urologic service complaining of pain which had been present since 1944 in the anatomical location of the right kidney. On July 10, 1950, this pain was worse and there was associated nocturia and dysuria. The admission hemoglobin was 14.0 Gm. per 100 cc. and a leukocyte count of 7,300 per cu. mm. was reported. On July 14, 1950 while in the hospital, he developed a different pain which began at the umbilicus and shifted to the right

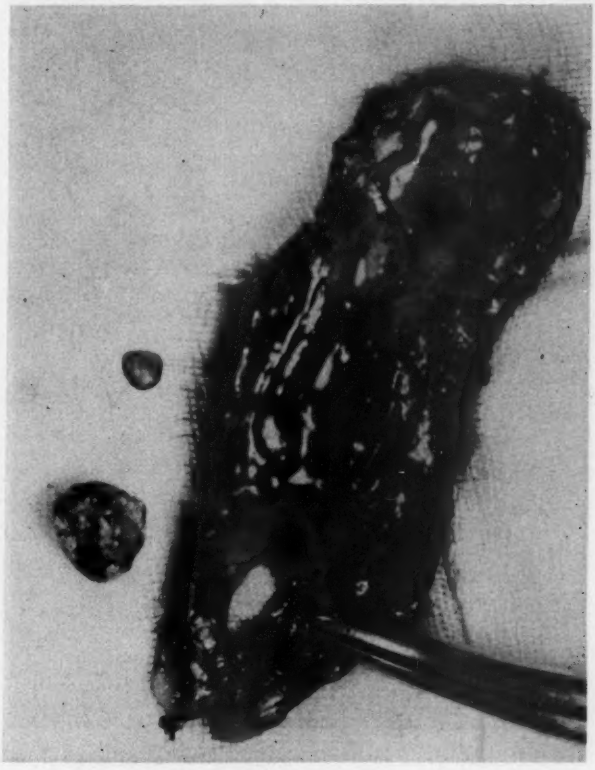

FIG. 3. Case 2. Photograph of operative specimen

lower quadrant of the abdomen. The leukocyte count promptly increased to 20,500 per cu. mm. and his temperature rose to 102 F. The urinalysis, including a microscopic examination, was entirely normal. When seen in the general surgical section, he had tenderness in the right lower quadrant, right flank and in the right kidney area. A plain roentgenogram was made as an emergency procedure and a clinical diagnosis was made of appendiceal calculi with perforation. Two calculi were present. Emergency retrograde pyelograms demonstrated that the calculi were not in the urinary tract (fig. 2). Exploration was promptly done, using a McBurney incision, and a gangrenous appendix with perforation and two calculi were removed (fig. 3). The calculi were 1.5 cm. and 0.4 cm. in diameter, respectively. The patient had an uneventful convalescence.

Case 3. R. P., a Negro male, age 56 years was admitted to the hospital on Oct. 15, 1951, complaining of pain in the right lower quadrant of the abdomen, which began on Oct.

12, 1951. He had localized tenderness at McBurney's point with muscle spasm. The hemoglobin was 13.5 Gm. per 100 cc. and the leukocyte count was 14,950 per cu. mm. The urine contained albumin, 10 to 15 polymorphonuclear leukocytes per highpower-field and an occasional erythrocyte, and was negative for sugar. A plain roentgenogram (fig. 4) of the

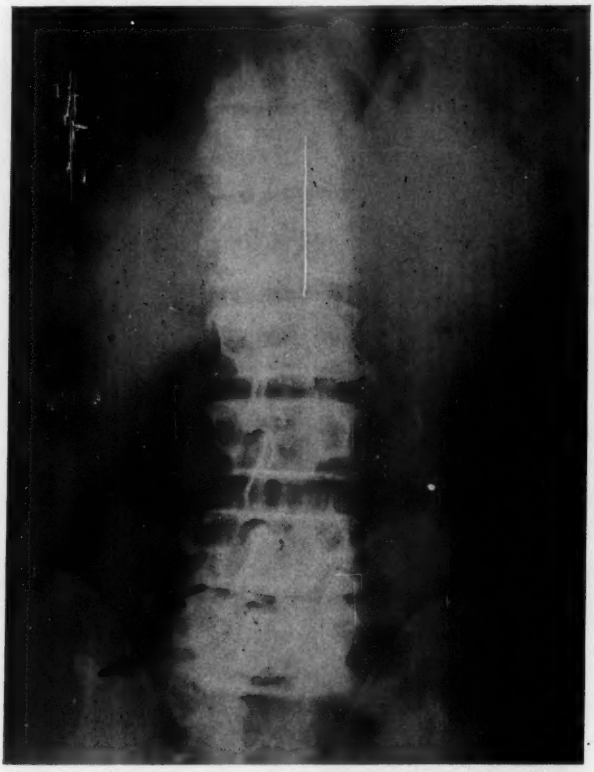

FIG. 4. Case 3. Roentgenogram showing two appendiceal calculi, one of which simulated a metallic screw.

abdomen showed two bodies, one resembling a screw. A clinical diagnosis of appendiceal calculi (one calculus of peculiar shape) with perforation was made. Using a McBurney incision, exploration was promptly done, and a gangrenous, perforated appendix containing two calculi (one simulating a screw) was removed (fig. 5). This patient developed a fecal fistula which was excised successfully at a subsequent operation.

Felson and Bernard[8] have suggested that the incidence of severe acute appendicitis when calculi are present approaches 100 per cent and that perforation exists in almost 50 per cent. All of our cases had perforated. Because of the high incidence of a severe form of appendicitis in calculus disease of the appendix, it has been suggested[1, 8] that if an appendiceal calculus is demonstrated by a roentgenogram taken for any cause, such an appendix should be promptly

excised. We agree with that concept. Pilcher[16] reported 1 case of an appendiceal calculus which was relatively asymptomatic.

FIG. 5. Case 3. Photograph of the operative specimen

CONCLUSIONS

1. Appendiceal calculi while uncommon, may not be so rare as has been indicated. They probably would be seen more frequently if plain roentgenograms of the abdomen were taken in all cases of active appendicitis.

2. The prevalence of appendiceal calculi is emphasized. They must be considered in the differential diagnosis of calcified masses, especially of the right lower quadrant of the abdomen.

3. Appendiceal calculi produce, most usually, a severe type of appendicitis.

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THE BROWN ELECTRODERMATOME AN INVALUABLE AID IN THE CARE OF MAJOR BURNS

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Split skin grafts, since first described by Ollier-Thiersch, have become the accepted method of treating burns by surgeons engaged in resurfacing skin defects. In the past their use, in the hands of the average surgeon, has been limited by the operator's skill in handling available instruments for procuring grafts. The general surgeon doing an occasional graft has found the cutting of a long strip of skin, of uniform width and depth freehand, as advocated by Brown, almost impossible. Certain modifications of the Brown knife, notably the Marck's thickness calibrating device made the method more practical, but still left much to be desired.

The Padgett dermatome, introduced in 1937 and later modified by others, made extensive split skin grafting more practical, but still required special skill on the part of the operator to obtain full drums of skin of a depth which would permit primary healing of the donor site. Other factors, independent of the operator's ability, such as humidity, excessive skin moisture or consistency of the glue used on the drum might lead to failure. Finally, the extent of available skin for grafting was limited by the availability of suitable donor sites, since many areas of the body do not lend themselves well to the use of the Padgett dermatome.

An instrument was introduced in 1947 by its inventor, Dr. Harry M. Brown, of Indianapolis, which is an improvement upon previous instruments used for skin grafting. This ingenious device was conceived by Dr. Brown while he was a prisoner of war in the Philippine Islands and developed after his liberation and return to this country. Dr. Brown's untimely death in 1948 prevented his realizing the full measure of the value of his invention. Its use has become widespread in the past three years and it is difficult to understand why no extensive reports of its value have appeared in the literature. We have been able to locate but three reports on the electrodermatome up to the present time.[1, 2, 3] The first was an abstract of a film made by Dr. Brown, the second was a description of the mechanical advantages of the machine, and the third was a report by Pratt of its use in 12 cases of lymphedema of the extremities. We believe that our experience with the electrodermatome should be published to demonstrate its versality and ease of operation, and to offer a technic for the handling of large grafts procured with the instrument.

Since previous publications have accurately and fully described the construction and principle of the electrodermatome, no discussion of this phase of the subject will be made here. In the past two and one-half years we have used the Brown electrodermatome 194 times. This number includes its use for

debridement of burn sloughs and phlebitic ulcers, as will be described later. Results have been uniformly good.

CUTTING THE GRAFT. The electrodermatome obviates the necessity of using cement. The donor site is prepared as for any surgical operation, then covered with a film of sterile mineral oil. The moving parts of the instrument are lubricated lightly with the same material. The operator maintains countertraction on the skin with a sponge held in his left hand. The instrument is set firmly against the skin at about a 20 degree angle to the surface. As soon as the motor is started the dermatome is pushed forward at a speed of about an inch every four seconds. As the graft feeds across the blade it is picked up with a hemostat by an assistant and held loosely to keep it away from moving parts of the machine. Tension on the strip of skin will produce a graft of uneven thickness and this must be avoided. When the end of the cut is reached, the handle of the dermatome is depressed, thus raising the blade from the skin. Each piece of skin is immediately placed in physiologic sodium chloride solution soaked gauze to prevent drying. Succeeding cuts are made immediately contiguous to the first. Thus an entire extremity can be utilized as donor area without difficulty and with a minimum of operating time. There is little danger of cutting grafts of a thickness that will prevent primary healing of the donor site if machine settings of .004 to .010 inches are used. The donor site is dressed with fine mesh vaseline gauze applied without pressure. This is covered with an elastic gauze dressing (kerlix)* applied with very gentle pressure.

In our experience the thigh and leg furnish the most satisfactory donor sites. Practically all areas of the body can be used, however, after acquiring some facility with the machine. Areas of the body with depressed surfaces and bony prominences, especially in emaciated patients, can be leveled off by rapid subcutaneous injection of an adequate quantity of physiologic sodium chloride solution. This maneuver, developed early in our experience with the machine (H. E. D.) is greatly facilitated by the use of needles 8 inches long with a no. 17 gage bore.

It has been our practice, on cases with defects which could be covered easily at one sitting, to deliberately cut 25 to 50 per cent more skin than was needed. This excess of skin is wrapped in sodium chloride solution soaked gauze and covered with vaseline gauze to prevent drying and is preserved in a refrigerator. When the first dressing of the graft is done, any loss can be replaced by the *banked* skin, thus obviating a second surgical procedure. No increase in morbidity has been noted as a result of this practice.

APPLICATION OF THE GRAFT. Due to the thinness of grafts which can be cut with the electrodermatome and to the *feathering* of the edges we have found that these grafts can be teased into position and will quickly become agglutinated to the recipient area. This eliminates the laborious task of suturing the skin in place. This placement of the graft is best done by a team of two working on the same strip of skin. Small curved hemostats are very satisfactory instruments for this procedure. Wherever the contour of the defect prevents accurate edge to

* Curity.

edge application of the skin, the overlapping skin is trimmed off. The grafts are *snubbed* into position with wet dressings of fine mesh gauze, covered with wrappings of kerlix. We have used an aqueous solution of zephiran in a $\frac{1}{5000}$, strength for soaking these dressings. Wherever the grafts are applied to areas subject to much motion, that part is immobilized by plaster or metal splints. When feasible an extremity is immobilized in a position of function.

AFTER CARE OF THE GRAFT. To keep dressings on the graft area wet, or to allow them to remain dry after the initial dressing is a decision which must be made to fit each individual case. In general, if the defect is a clean surgical wound, the dressings are permitted to dry, whereas on contaminated areas they are kept wet to promote drainage.

The dressings are usually changed on the fifth postoperative day unless a febrile reaction indicates the need for earlier dressing or difficulty or reimmobilization should make it advantageous to leave the primary dressing in place for a longer time. We have routinely used light anesthesia for dressings in children, since crying and movement might disturb the graft and produce unnecessary

TABLE I

No. Cases	Area Covered	Graft Loss
35	Less than 20 sq. in.	All over 80% take
66	20-80 sq. in.	Two complete loss One 50% loss
27	80-150 sq. in.	One 25% loss
48	Over 150 sq. in.	Two 25% losses

loss. At the time of the first dressing, hematomas or serum collections may be evacuated, and any necrotic tissue may be trimmed.

When the graft is completely successful it is redressed with fine mesh vaseline gauze and kerlix. Splints are replaced, and if the graft crosses a joint, immobilization is maintained for from two to three weeks. If additional granulating areas remain, or if the graft is not completely successful, the wet dressings are continued for two to five days longer.

While the electrodermatome finds its greatest use in resurfacing burns, it is also valuable for grafting avulsed wounds, stasis ulcers, defects after radical mastectomy, and for many other lesions.

The electrodermatome has been used 194 times by us during the past two and one-half years. Of this number, it has been used 176 times for autografts, 7 times for donor or homografts, and 11 times for debridement of sloughing tissue. Of the 176 autografts only two were complete losses, both of these being less than 40 square inches in area. One result is listed as 50 per cent loss and three results as approximately 25 per cent losses. All others were estimated as 85 to 100 per cent takes. In 6 patients large areas ranging in size from 350 to 450 square inches were grafted with 90 to 100 per cent success. The areas covered by autografts and the approximate graft loss is shown in Table I.

Seven homografts have been taken with the electrodermatome, each being between 200 and 400 square inches in area. All have been uniformly successful in improving the general condition of the patient and preparing him for autografts. The life of the homografts may vary from 2 to 10 weeks, after which they melt away completely.

Debridement with the electrodermatome has been utilized 11 times, usually to speed the separation of burn eschar and permit earlier grafting. In 4 cases debridement was followed by immediate grafting. No graft loss was noted in any case. The electrodermatome is set for .025 to .040 inch thickness and the eschar is removed in strips, down to healthy tissue. If skin is not to be applied immedi-

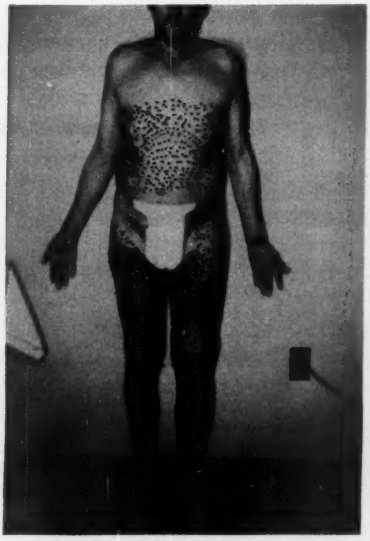

FIG. 1

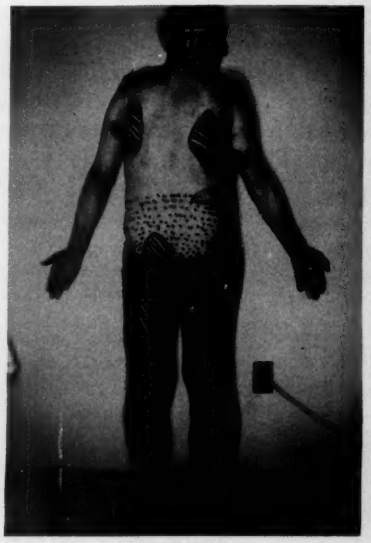

FIG. 2

FIGS. 1 and 2. Cross hatched area represents third degree burns, stippling indicates second degree area.

ately, the denuded area is carefully dressed with a fine mesh gauze (kerlix) dressing and soaked with $\frac{1}{5000}$, aqueous solution of zephiran.

A single case report is presented to emphasize the value of the electrodermatome in the management of extensive burns.

CASE REPORT

S. H., a 44 year old white man, was admitted to the hospital on Dec. 31, 1950, approximately two hours after being burned in an oil well explosion. On admission to the hospital he was found to be in moderately severe shock and suffering from third degree burns covering approximately 40 per cent of his body surface, second degree burns over 20 per cent of the body, and a compound dislocation of the left elbow. His burns were dressed with fine mesh gauze and kerlix and the burned extremities were immobilized with plaster splints. The dislocated elbow was reduced and immobilized. His general condition improved for a

short time and he began taking adequate oral fluids. About eight hours later he vomited and went into profound shock. A cannula was placed in an ankle vein and transfusions, followed by suitable fluids, were given. He again improved and his urinary output rose to normal. The following week was uneventful and there was no further vomiting. The temperature curve ranged from 100 to 101 F. taken by rectum. Blood chemistry was checked and found to be within normal limits. On Jan. 9, 1951 he began hiccoughing and shortly afterward vomited. Intravenous therapy was resumed. The following day ACTH was administered in doses of 15 mg. every 6 hours and the hiccoughing and vomiting ceased. He was irrational, but his mental condition cleared within eight hours after the first dose of ACTH. On January 19 he was taken to the operating room and under light anesthesia the original dressings were removed. The slough on the third degree areas was found to be hard and leathery and incompletely separated. The second degree burns were well healed. In view of the general

FIG. 3

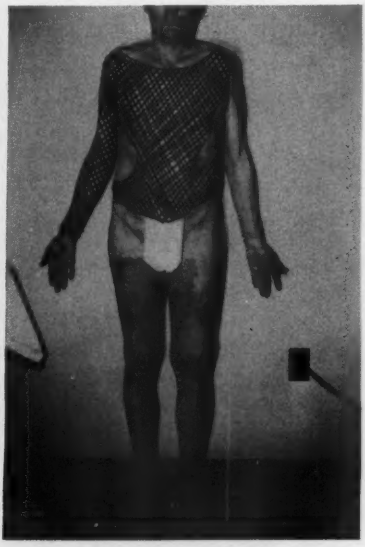

FIG. 4

FIGS. 3 and 4. Cross hatched areas indicate extent of electrodermatome donor sites

condition of the patient and the fact that some delay was necessary before the healed second degree areas could be used as donor sites, it was decided not to debride the eschar with the electrodermatome. Dressings of aqueous zephiran solution were applied and changed daily for the next nine days. On February 1, after the usual preparation, his chest was *contoured* by the rapid subcutaneous administration of 2000 cc. of physiologic saline solution. This area was then used as a donor site to remove about 280 square inches of skin with the electrodermatome set at .010 inches. The skin obtained was used to resurface the circular defect around the left ankle, the left calf and the left thigh. The grafts were placed and the leg splinted. On February 5 the graft was dressed and found to be successful. On February 6 his burns were redressed and the following day the unburned areas on the abdomen and in the flanks, and the entire unburned portion of the right arm were used as donor sites for about 350 square inches of skin taken with the electrodermatome set at .010 inches. This skin was used to resurface the right thigh and leg burns. Dressings on February 11 showed this graft to be 100 per cent *take*. The remaining burned areas were redressed on the following day and

on February 13, using subcutaneous sodium chloride solution for *contouring*, about 200 square inches of skin was removed from his back and used to resurface the burns on his right arm, both shoulders, buttocks and the upper portion of both thighs posteriorly. The patient was placed on a Stryker frame, face down, for the operation and was returned to his room in the same position. Graft dressings on February 17 revealed that he was completely healed except for a few marginal granulations. Vaseline gauze dressings were placed on all grafts on February 18 and he was allowed to lie in the Fowler's position. One week

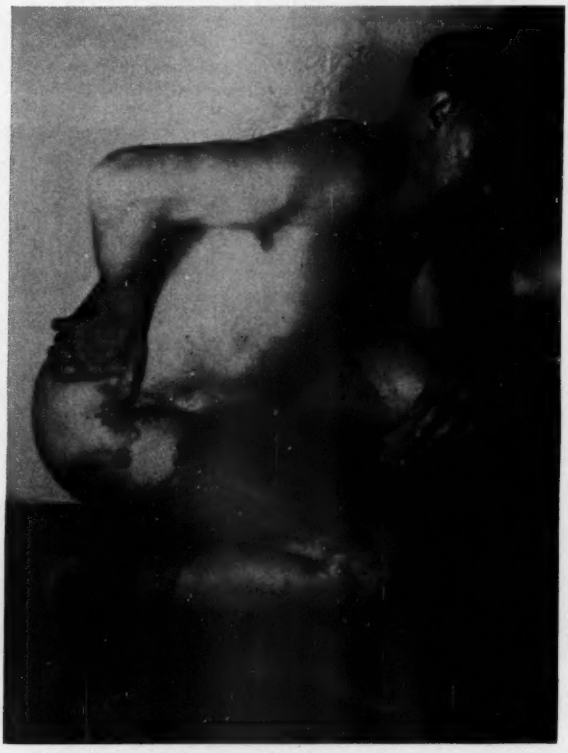

FIG. 5. Photograph of patient nine months after burns demonstrating return to normal function.

was allowed for final consolidation of the latest graft and he was discharged on the fifty-eighth hospital day completely healed. The associated compound dislocation of the left elbow had healed satisfactorily and the only residual disability was a partial ulnar paralysis due to traction on the nerve at the time of injury.

CONCLUSIONS

The Brown electrodermatome is a practical, efficient instrument for procuring split skin grafts of uniform thickness and width. The area of skin which can be taken is limited only by the undamaged skin on the patient's body.

A technic has been described for the application and after care of skin grafts which has proved satisfactory in our hands.

A case report has been presented to demonstrate the usefulness of the electrodermatome in extensive burns.

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ANTIBACTERIAL AGENTS IN EXPERIMENTAL STRANGULATION OBSTRUCTION

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Antibiotic agents have reawakened interest in the role of bacteria in the fatal outcome of intestinal obstruction with strangulation. In our previous studies[6, 7] of experimental strangulation obstruction we have found that attention to fluids, electrolyte balance, and blood replacement has significantly prolonged survival. More recently the exotoxins of Clostridium welchii have been shown to be one of the major lethal factors in acute intestinal obstruction.[3]

A study was made to evaluate the effect of preoperative bowel sterilization and postoperative antibiotic therapy on the survival of dogs with strangulation obstruction.*

The work was divided into two sections:

1. (a) Preoperative preparation with streptomycin and sulfathaladine;
   (b) postoperative treatment with penicillin and intravenous aureomycin.
2. (a) Preoperative preparation with oral aureomycin;
   (b) postoperative treatment with penicillin and intravenous aureomycin.

In each group we followed the clinical picture, bacteriology and enzymes of peritoneal fluid and bowel contents, white cell counts and specific gravity of the blood and peritoneal fluid, and electrolytes.

METHODS. During the preoperative period, which varied from three to six days, only water and drugs were allowed by mouth. Intravenous fluids maintained nutrition.

At operation, performed according to a previously described technic,[6] all veins to a 30 cm. segment of the small bowel were divided. The bowel was obstructed by dividing it and inverting the two ends 135 to 150 cm. distal to the ligament of Treitz. Peritoneal fluid was aspirated through latex tubes at intervals during the survival period. The animals were observed continously until death, when autopsy was done immediately. Studies were made at regular intervals.

RESULTS. Fourteen dogs were operated upon. Nine were prepared with oral streptomycin and sulfathaladine. Five were prepared with oral aureomycin. The two groups will be discussed together since there seems to be no essential difference in the results.

Five dogs were excluded because of death from extraneous causes as follows:

1. Death at 10 hours due to hemorrhagic shock and failure to respond from anesthesia.

From the Department of Surgery, Louisiana State University School of Medicine, New Orleans, La.

Paper delivered during a meeting of the Louisiana Chapter of The American College of Surgeons, October 24, 1952, New Orleans.

* The experimental work described in this paper was done at the Harrison Department of Surgical Research, School of Medicine, University of Pennsylvania, in collaboration with Drs. H. R. Hawthorne and A. Gelb.

2. Death at 29½ hours, while receiving intravenous potassium chloride rapidly, due to hyperpotassemia.
3. Death at 25 hours due to perforation of bowel.
4. Death at 38 hours due to peritonitis from perforation of bowel.
5. Death at 33½ hours with 800 cc. almost pure blood in the peritoneum and attributed to shock.

Nine dogs survived from 28½ hours to 116 hours, with an average survival time of 71½ hours. This is twice the average time of our series without antibiotics.

The usual postmortem findings were as follows: The strangulated segment of bowel was a dark purplish black and was sharply demarcated at each end from adjacent normal bowel by both its color and thickness. Both mesenteric and antimesenteric sides of the bowel were thickened. The mesentery of the strangulated segment was thick, hemorrhagic and edematous to the point of ligation of the vessels. The mucosa was red to black, but was not completely destroyed. There was little change in the remainder of the viscera.

Additional conditions which hastened death were as follows:

1. Angulation of the bowel, with a closed loop obstruction, in two experiments.
2. Intussusception in two experiments.
3. Perforation in the viable bowel in one experiment.
4. A questionable leak in the strangulated segment and massive consolidation of the lung in one experiment.

WHITE CELL COUNTS. The white cell counts in the peritoneal fluid showed a uniform upward trend in marked contrast to previous findings.[3] The white cell count of the blood showed the same general trend.

In these experiments the white cell count of the blood was almost always higher than the white cell count of the peritoneal fluid in contrast to the opposite findings in our previous work. Furthermore, the peak values here were much lower than they were in the previous series.[3]

PERITONEAL FLUID. In the series without antibiotics there were changes in the peritoneal fluid late in the course of each experiment.[6, 7] The late fluid was malodorous, darker in color, and the red cells were hemolysed. After centrifugation there was a definite difference between the early and late fluids. In the present series, these changes usually did not occur, and could not be correlated with the clinical course, as in the previous series. The foul odor was not uniformly present. Change in color rarely occurred. Hemolysis after centrifugation was not significant. A new pigment, found by spectrophotometric examination in the peritoneal fluid of the previous series, was not uniformly found in the present group, nor could its presence be correlated with the remainder of the clinical picture.

FLUIDS. Fluids were analysed on a basis of cc. per Kg. of body weight per 24 hours. No correlation could be found between survival and intake of any of the components.

BACTERIOLOGY. The bowel lumen was essentially sterile at operation. In the peritoneal fluid, Pseudomonas aeruginosa was found in three experiments, and

the Clostridia were found in two having small perforations. The bacteriologic findings of the peritoneal fluid could not be correlated with any other findings.

ENZYMES. Because of our interest in the role of Clostridium welchii in strangulation obstruction, and because others have shown that the alpha toxin of Clostridium welchii may be demonstrated in strangulation obstruction,[1] we used the lecithinase turbidity test on the peritoneal fluid and bowel contents. Lecithinase activity could not be detected in this series.

MICROSCOPIC PATHOLOGY. The histologic changes in the strangulated bowel are in striking contrast to those from dogs not receiving antibiotics.[6] In every case the bowel wall was greatly thickened with hemorrhage and edema, the hemorrhage being most marked in the muscularis and next most prominent in the submucosal layer. The mesentery was likewise thickened. The normal architecture was usually well preserved, each of the various layers being distinct, and in good contion. In some cases the mucosal layer appeared almost normal. This preservation of structure is in severe contrast to the complete destruction in the nonantibiotic series.

DISCUSSION. Preoperative sterilization of the gastrointestinal tract combined with postoperative antibiotics in large doses have significantly prolonged survival in experimental bowel strangulation obstruction. Originally we were confused by similar results obtained by Blain and Kennedy[2] using postoperative penicillin as the only antibiotic. Recently, however, Lepper, et al[4, 5] have shown that intravenous aureomycin in doses of over 40 mg. per Kg. of body weight per 24 hours can be toxic, and Schweinberg, at al[9] have shown that this dose can nullify the defense mechanisms of animals with peritonitis. Unfortunately, all of our animals received aureomycin in the toxic range. We now believe that some previously unexplained deaths were due to excessive intravenous aureomycin. We are presently trying to repeat these experiments using other routes of administration of aureomycin in the hope that indefinite prolongation of life will be possible. Rabinovici and Fine's[8] demonstration of the effectiveness of oral aureomycin in bowel strangulation without obstruction prompts us to believe that similar results might be possible in strangulation obstruction.

CONCLUSIONS

1. Preoperative oral antibiotics effectively sterilize the bowel of dogs.
2. Strangulation obstruction may be created and studied after this type of preparation in order to further evaluate the bacterial factors in strangulation obstruction.
3. Intravenous aureomycin may be toxic, and may have contributed to the death of our experimental animals.
4. The white cell count of both the blood and the peritoneal fluid showed a continued rise in these experiments, in contrast to a significant terminal drop in similar animals not treated with antibiotics.
5. Lecithinase was not detected in either the peritoneal fluid or bowel contents in these experiments.
6. The histologic appearance of the strangulated segment showed marked

preservation of structural detail in contrast to the destruction in nonantibiotic experiments.

7. The survival time of dogs with adequate preoperative bowel sterilization and postoperative antibiotic treatment was significantly prolonged over that of dogs not treated with antibiotics.

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INJURY TO THE GENITOURINARY TRACT DURING SURGERY

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Because of the rather extensive number of reports and articles given over to the various methods of treatment of injuries to the urinary tract during surgery, it is reasonable to assume that this complication is not as uncommon as it might seem. During the past 15 years there have been approximately 78 papers devoted to this particular subject. It is understandable and natural that a great many such injuries would be difficult to foresee in view of some of the radical surgery now carried out since the advent of improved surgical technics, antibiotics, etc.

As complications of pelvic surgery may occur during the operation, or may develop later as a result of scar formation, any injury to the ureter, immediate or delayed, is a serious complication in surgery. Injuries occurring during operation, are more frequent than are late sequelae and may involve one ureter, both ureters, the bladder, and particularly in vaginal surgery, the base of the bladder. By far the largest percentage of complications result from gynecologic operations, but also exist in other types of radical pelvic surgery and in urologic surgery.

The exact type of injury may be difficult to determine unless recognized immediately at the time of operation. Regardless of the recognition, treatment is difficult and the results of any immediate repair difficult to evaluate for some time. The types of injuries to the ureters are quite varied. A ureter may be included in a ligature, and the lumen partially occluded with resultant distortion of the remainder of that portion of the ureter, or it may be the so called *parietal injury* in which part of the external portion of the ureter may be included in the ligature, or crushed with a clamp with a resultant necrosis of the ureter and escape of urine within a short period of time.

Of somewhat less significance, but sufficiently serious, are injuries to the bladder. Of these injuries, probably the most common is direct incision into the bladder cavity followed, too frequently, by inclusion of part or all of the bladder wall in sutures, or interference with the nerve and blood supply to the posterior aspect of the bladder wall by clamping or ligating which does not infringe directly upon any proper layer of the bladder.

Prior to the advent of more radical surgery, vesicovaginal fistulas were few and usually the result of obstetrical procedures. But, in recent years, the percentage of vesical injuries is rising. The location of a large portion of vaginal fistulas following vaginal surgery makes both prevention and repair equally difficult. They are usually found high in the vaginal vault and, upon cystoscopic examination, the vesical opening may be found on the upper level of the trigone or just above. Approximately 1 cm. of the bladder wall in this area is in extremely

Presented during the annual assembly of The Southeastern Surgical Congress, March 10–13, 1952, Atlanta, Georgia.

close contact with the anterior vaginal wall and it is in this same area that most injuries to the bladder occur during vaginal surgery.

A great many injuries are found to occur in cases in which previous pelvic or lower abdominal surgery has been done. It is very easy to imagine that the bladder can become firmly and highly adherent on the anterior abdominal wall and can be very easily entered if it is distorted. Large masses in the pelvis such as uterine fibroids, large cysts, etc. may change the position of the viscus laterally to an abnormal location. In addition, further immediate injuries to the bladder may come as a result of vaginal surgery; either in plastic work on the anterior vaginal wall, a high amputation of the cervix, vaginal hysterectomy, and in interposition operations. Widespread carcinoma of the fundus of the uterus with fixation, marked chronic inflammatory reaction extending into the adnexa, ovarian malignancies, etc. all displace the normal pelvic relationships.

In the prevention of these complications the same factors hold true for all surgery whether it be gynecologic, abdominal perineal section, or urologic. Fundamentally, the surgeon should have a thorough knowledge of the pelvic organs including the bladder and lower ureters; all of their possible anomalies and possible variations from normal due to previous surgery, inflammatory or neoplastic masses. To be properly prepared in this respect, several things may be of valuable assistance. Especially in patients who have undergone previous major abdominal or pelvic surgery, an adequate survey of the genitourinary tract should be made. This should include at least adequate excretory urograms and, if at all indicated, cystoscopy. This not only gives an adequate survey of the normal urinary tract with definition of any anomalies such as reduplicated ureters, Y ureters, etc., but also will give a clue as to whether or not there exists any damage from previous disease or surgery. Real value should be gained from cystoscopy performed for evaluation, with special reference to patients who have had multiple pelvic operations.

We are all only too familiar with the small, distorted and/or contracted bladder which is the constant complaint of a great many women who have undergone repeated pelvic surgery. Very frequently, minor urologic conditions disclosed by such cystoscopy may be remedied at the time major surgery is undertaken and, at least, serve as a guide for future care of any bladder pathology which may develop following surgery.

Following such preventive steps, the other factors which are so necessary to good surgery all come into place. Good and adequate anesthesia, satisfactory exposure of the operative field, skilled assistance, extreme care and a constant knowledge of all the predisposing factors are all of material aid in reducing complications involving the urinary tract. As previously mentioned, the proximity of the upper portion of the anterior vaginal wall and the posterior aspect of the bladder encourages the formation of a vesicovaginal fistula. The same close relationship of tissue structures probably accounts for a majority of lower ureteral injuries.

The ureter passes posterior to the uterine artery which crosses the ureter at about 1 inch above the entrance to the bladder. Thus it is that in secondary

clamping of this vessel for bleeding, a clamp placed in a slightly lateral position may catch the ureter. The same is true in the placement of deep sutures. Nor is all the danger eliminated here by using the vaginal approach. For in procidentia the ureters frequently may be drawn down with the prolapsing uterus and, in this abnormal position, become quite liable to injury. With this in mind, placement of ureteral catheters under anesthesia would seem to be a definite help especially in cases where previous operative procedures have been done, or where a known and suspected deviation from the normal urinary tract is a consideration. Although this is done routinely by some operators, some consider this of questionable merit as the catheter is frequently not palpable within the ureter at the time of operation.

Immediate repair should be done, if possible, with the procedure of choice lying between ureteroureteral anastomosis and reimplantation into the bladder. Either may be considered if the division of the ureter is sufficiently low. However, if the division of the ureter is too high to permit implantation into the mobile portion of the bladder, then an end to end anastomosis is desirable, using a ureteral catheter as a splint. At no time should any noticeable stress be placed on a ureter that has been reimplanted because it will merely retract and leave a draining fistula. If it is impossible to repair or implant the ureter, ligation is indicated and, if there is no infection the kidney atrophies in a great many cases and further procedure is unnecessary. Knowledge of the status of the contralateral kidney is invaluable since mere palpation is insufficient to determine single renal sufficiency.

It is possible for both ureters to be ligated, in which case the patient will be anuric; catheterization will yield no urine, and cystoscopy and ureteral catheterization are imperative. If obstructions are met by ureteral catheters bilaterally, the condition of the patient should be improved as quickly as possible, the abdomen opened, the ligated or sectioned ureters identified, and ureteral catheters passed to both renal pelves and allowed to remain for approximately 72 hours. It may be possible in an occasional case for a catheter to be passed through a loosely tied ligature but this is extremely rare. Manipulation of the catheters from below, after the abdomen has been opened, facilitates identification and deligation for repair of the damaged ureters. Nephrostomy should be used as a life saving measure under local infiltration if the condition of the patient does not permit reopening of the abdominal wound.

In the late treatment coincident to bladder injuries, vesicovaginal fistulas which do not respond immediately to a combination of postural drainage, indwelling catheter, and antibiotics, usually respond best to transvesical repair. The vaginal approach is likewise very unsatisfactory to close a ureterovaginal fistula. In these cases, if a ureter can be catheterized at all, gradual dilatation will frequently cure the patient. Likewise, it is not too unusual to have spontaneous closure of a uretero-cutaneous fistula following catheter drainage and adequate dilatations.

For ureteral injuries, late operations such as end to end anastomosis or ureteral implantation into the urinary bladder are seldom successful. Recently we have repaired one of the latter adequately due to low section of the ureter and an

easily mobilized ureter with high implantation into the dome of the bladder. Uretero-colic anastomosis must be considered for preservation of needed renal function, as must nephrostomy in some cases. Nephrectomy is done for the resulting hydro-ureter and hydronephrosis if atrophy does not take place and satisfactory secondary repair is impossible.

It is not to be supposed that all surgical injury to the genitourinary tract is a result of gynecologic surgery, for we must also consider extensive operative procedures for carcinoma of the sigmoid and rectum, the occasional complication from the vermiform appendix, and the inclusion of a portion of the bladder or ureter, or both, in the inguinal hernia sac during repair. Too, diverticulectomy or resection of the bladder wall for carcinoma may also result in severe ureteral damage.

We thus are able to recognize that with the increasing importance of more radical surgery, and with the drugs, skill, and equipment at hand to do this surgery we may find that the accidents discussed are difficult to prevent. Yet the usual array of injuries would undoubtedly include some that are preventable. Adequate preoperative work-up of the urinary tract, including excretory urograms and cystoscopy, would seem advisable. There is certainly nothing to be lost by immediate preoperative ureteral catheterization in selected cases.

Keeping in mind the same principles which apply to all good surgery, this work-up and preventive effort should facilitate the surgery, considerably ease the more difficult procedure, permit more easily the immediate recognition of injuries, and with this recognition of the injury allow more adequate, knowledgeable, and prompt repair with less resultant total damage.

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THE DIAGNOSIS AND TREATMENT OF NECK, SHOULDER AND ARM PAIN

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The syndrome of intractable types of pain in the neck, shoulder and arm occurs with sufficient frequency to be one of the more important diagnostic problems that the surgeon sees. These syndromes have been ascribed, at times erroneously, to brachial plexus neuritis, bursitis, arthritis, myositis, fibrositis, and other conditions. It is the purpose of this paper to summarize the pathological physiology of these syndromes and describe some methods of treatment.

For decades surgeons have realized that pain is due to irritation of nerves or pressure on nerves, and that, although this pain may be referred by sympathetic pathways, the usual cause of the pain lies in the pathological stimulation of sensory nerves. The most frequent causes of nerve irritation are the scalenus anticus syndrome, osteoarthritis of the cervical spine, and lesions of the cervical intervertebral disks. Other conditions exist, but the signs and symptoms in the latter are usually much more localized than they are in the former conditions. In Table 1 the more common conditions which must be ruled out are listed.

The scapulocostal syndrome[10] can usually be ruled out by the location of pain in the posterosuperior aspect of the shoulder girdle, often developing in people with poor posture who work over a desk or a typewriter all day. There is usually present a trigger-point tenderness in the rhomboid muscle at the upper medial border of the scapula.

The shoulder-hand syndrome and Sudeck's atrpohy[12] are usually associated with sympathetic or trophic disturbances, and most often follow trauma. Local procaine infiltration of the tender area, or sympathetic block in the early stages will produce immediate relief of the pain and sympathetic changes which consist of cyanosis, increased sweating or edema.

Tumors located in the medial supraclavicular area are rare,[1] but when present they may represent a metastasis from carcinoma of the stomach, a Pancoast tumor, or a pulsating aneurysm. The signs and symptoms are due to brachial plexus compression, and a large percentage have a Horner's syndrome secondary to involvement of the sympathetic chain.

Cardiac disease may often be confused with a cervical disk,[5, 8, 12] and in our experience this has occurred sufficiently often to bring it to the attention of physicians in general. About 10 per cent of the cervical disks on the left side produce severe pain in the anterior and left sternal region, due to referred pain over the fourth cervical dermatome. The presenting symptoms may be a cardiac type of pain. This should always be considered in the age group of 40 to 60 years, when the electrocardiogram is normal and no definite evidence of angina pectoris

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Presented during the annual assembly of The Southwestern Surgical Congress in Dallas, Texas, October 20–22, 1952.

is found. In such patients the apparent cardiac pain may be reproduced by lateral flexion of the neck to the left with mild pressure on the vertex (the so-called Spuring test).

Brachial neuritis or meningoneuritis may present a difficult diagnostic problem.[9] A history of arm injury with traction on the plexus will usually confirm the diagnosis of the traumatic type, but the insidious infectious type with an irrelevant history may lead to a faulty diagnosis. In our experience the infectious type is relatively uncommon and very rarely occurs without obvious signs of generalized toxemia or involvement of other nerves or muscles in the body.

Subdeltoid bursitis and localized periarthritis of the shoulder are almost always diagnosed by extreme limitation of motion at the shoulder with point tenderness. Both conditions react favorably to homolateral cervical sympathetic blocks and with physiotherapy or roentgenotherapy.

TABLE I

Conditions commonly confused with cervical disk

	Neck Pain	Upper Arm Pain	Hand Pain	Finger Numbness
Scapulocostal syndrome (trigger area)	+	+	+	+
Shoulder-hand syndrome Sudeck's atrophy (sympathetic changes)	0	+	+	+
Pancoast tumor aneurysms (palpable)	+	+	+	0
Cardiac disease (E.E.G.)	+	+	+	
Brachial neuritis, infectious traumatic (history)	+	+	+	+
Subdeltoid bursitis, periarthritis of shoulder girdle (trigger)	0	+	+	0
Cervical cord tumor or abscess	+	+	+	+

Cervical cord tumors[2, 3] or abscesses will show signs of cord compression, and the weakness of the involved muscles usually is much greater than in the other conditions. A slowly developing neurofibroma, however, may simulate very closely the cervical disk, as both cause pressure locally on individual nerve roots.

We now come to the main problem; *viz.* the differential diagnosis between the protruded or ruptured intervertebral disk of the cervical spine and the compression of the brachial plexus by a taut scalenus muscle, by a cervical rib or rarely by the pectoralis minor muscle in the costoclavicular syndrome. The answers to the fundamental differences are found essentially in the origin and location of the pain, the posture of the patient's head, the reflex changes, and the postural alteration of the radial pulse.

The following discussion is based on a study of 132 cases of cervical disks and 66 cases of scalenus anticus syndrome. The pain in both of these conditions may be confined to the shoulder, neck and arm area, but in the scalenus anticus syndrome there is very rarely fronto-occipital head radiation of the pain. The so-called anginal pain referred to the fourth cervical dermatome is rarely found in

the muscle syndromes. The distribution of the hand pain in the cervical disk tends to be more frequently in the index and middle fingers, while in the scalenus anticus syndrome the distribution of the pain tends to follow the ulnar nerve area, suggesting that cervical-7 and cervical-8 dermatome involvement is more common in the muscle compression syndromes, and that cervical-5 and cervical-6 involvement is more frequent with cervical spine pathology. Movement of the neck produces pain in both conditions, but rotation or flexion to the painful side may relieve the pain of the scalenus anticus syndrome. Likewise, the scalenus anticus pain is often increased by raising the arm, and the patients will say that the pain appears when they are washing dishes or driving a car or operating with their arms outstretched. In the scalenus anticus syndrome also, the pain and numbness is greater at night when the patient is in the prone position. The posture of the head may be important, as shown by Spurling and Walker,[11] who emphasized that in cervical disks the head tends to be held away from the side of the pain.

Other dissimilarities include the more frequent diminution or absence of the triceps reflex in the scalenus anticus syndrome, and alteration of the biceps reflex in cervical disks. The ulnar area of the hand is found to be hypesthetic or numb in the scalenus anticus syndrome, and the radial median nerve distribution is more often involved in the cervical disk.

The problem of osteoarthritis of the cervical spine, with or without cervical disk, must be mentioned. In both conditions we have a compression of the nerve root as it leaves the spinal canal, and it is here that the nerve root is often swollen and subjected to physical irritation by very minor changes in structure or position of the bones of the spine. The anteroposterior diameter of the intervertebral foramen is made smaller in both conditions. Consequently the pain is produced by movement. This is especially important in compensation cases where a neck injury has occurred and signs of a disk are found with the roentgenologic evidence of osteoarthritis. As pointed out by other observers, the two conditions may well coexist, and it is often impossible to differentiate these two pathological processes until the time of laminectomy.

In the scalenus anticus syndrome sympathetic changes are commonly found and rarely are present with the cervical disk. These consist of cyanosis, swelling of the hand, and increased sweating, and may in rare cases of long-standing require section of the sympathetic chain, as well as the scalenus anticus muscle. These sympathetic changes, as well as the pain, may be produced by abduction and external rotation of the arm or pressure over the belly of the scalenus anticus muscle. The production of symptoms by the pectoralis minor muscle has been described by Love[8] and may enter into the hyperabduction syndrome. The important feature to remember is the relative diffuseness of the arm and hand symptoms in the scalenus anticus syndrome, and the more sharply localized signs in the cervical disk pathology. Roentgenograms may be of definite benefit when we see in the scalenus anticus syndrome an enlarged transverse process or cervical rib, but this is relatively rare in these cases. In the cervical disk case we see narrowing of the intervertebral space, reversal of the normal spinal curvature

(fig. 1), and localized osteoarthritis (fig. 2) and in the important oblique roentgenograms narrowing of the intervertebral foramina. An aneurysm may be noted in the rare case of severe compression of the subclavian artery by the scalenus anticus muscle. Two cases have previously been described by our group and are believed to be caused by trauma to the artery following compression between the clavicle and first rib.[6]

The diagnosis is often made when we start therapy. Cervical traction is usually accompanied by relief of pain in the cervical disk after 48 hours, but may be

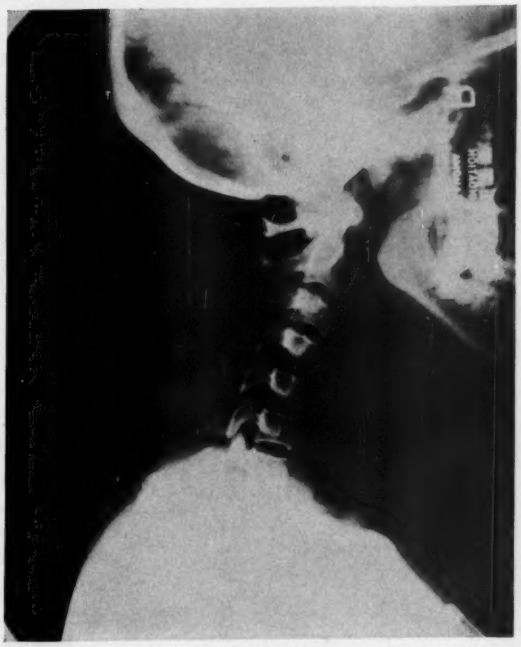

FIG. 1. Roentgenogram showing straightening of the cervical spine in an acute cervica disk.

increased in the scalenus anticus syndrome. Seven pounds of traction for 20 hours a day, is utilized with phenobarbital and codeine in ½ grain doses, three times a day after meals and at bedtime. Tolserol or other muscle relaxants should also be given during the period of traction, which should be continued for a period of 7 to 10 days.

I would like to discuss the type of traction we use and to briefly describe a new method of holding the head in proper position. The halter type of traction has not been very satisfactory due to the fact that the head is often hyperextended, and the patients complain of the pressure on the tip of the jaw. A new device, therefore, was made, utilizing steel wire, foam rubber and a nylon cover, in the form of a headgear which distributes the pressure to a much greater de-

gree. The accompanying illustrations show this apparatus (figs. 3 and 4). This apparatus is much more comfortable than the halter, and we are able to use many more pounds of traction for longer periods of time without discomfort.

In the cervical disk cases, in the event the patients have not improved after a long period of conservative treatment, it is sometimes necessary to use operative therapy. This was true in approximately 8 per cent of our series. The operation is usually performed through an extradural approach by making a small hole in the lamina. In large calcified disks or midline disks, it is necessary to do

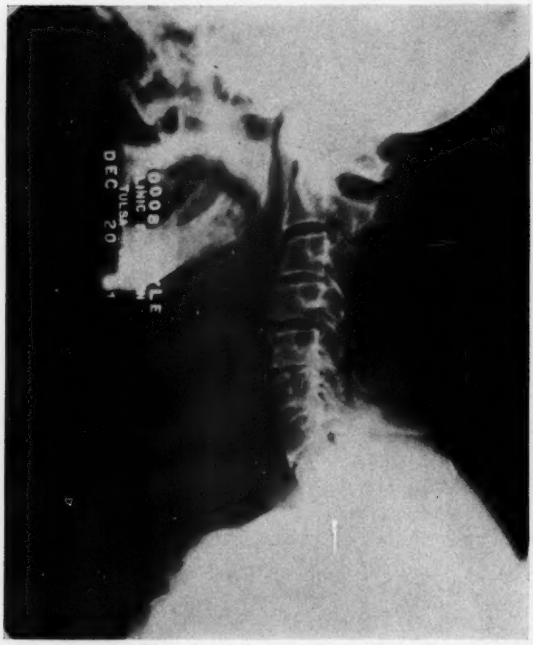

FIG. 2. Roentgenogram showing narrowing of fifth and sixth cervical interspaces with osteoarthritis in a cervical disk case.

a transdural approach and gently retract the cord to one or the other side. With small calcified disks a decompression of the nerve roots alone is the procedure of choice. Postoperatively there is occasionally a weakness of both lower extremities as well as temporary difficulty in urination. In cases with severe occipital headache we often extend the incision upward and section cervical-1, cervical-2 and the upper half of the cervical-3 dorsal roots to be certain the headache is alleviated. The cause of this upper cervical pain with a cervical-5 or cervical-6 disk is probably secondary to abnormal posture.

In the scalenus anticus syndrome it is necessary to remove a portion of the muscle, as there is a tendency for fibrous bands to develop postoperatively and mitigate the decompressive procedure. It is our policy to remove at least a 2 cm.

FIG. 3. "Head gear" type of cervical traction

FIG. 4. New traction apparatus in use

section of the muscle and to be certain that the most medial portion of the tendon is adequately removed. Likewise, more laterally some of the fibers of the scalenus medius are occasionally divided to permit a more thorough decompression of the plexus. The subclavian artery itself is rarely exposed at the time of surgical interference, except when an aneurysm is present.

In concluding the treatment of the scalenus anticus and cervical disk syndromes, it should be mentioned that procaine blocks of the scalenus muscle may often produce a relaxation and an alleviation of symptoms. Likewise, procaine injection in the occipital nerves will relieve much of the neck and head pain that may be present in cervical disks. This is a good diagnostic as well as a therapeutic procedure.

In regard to the treatment of osteoarthritis of the cervical spine, cervical traction is usually of temporary or partial benefit. On four occasions we have carried out a decompressive procedure with uncapping of the nerve root canal, and in 2 of these cases there was definite improvement in the pain, and in the other 2 very little alleviation of the symptoms.

The results of treatment of 132 cases of cervical disk is summarized as follows: 15 per cent obtained slight improvement by traction, 12 of this group had laminectomy with removal of the disk, 20 per cent had moderately good results with traction, and were able to carry on with their daily activities with a minimum of pain, while the remaining 65 per cent were able to return to their normal activities free from pain. Surgical procedures brought about improvement in all cases, but in 2 cases there was continuation of pain in the shoulder and arm to a lesser degree, and in 1 case weakness and numbness of the upper extremity. In none of this series did we have weakness or paralysis of the legs which probably follows acute flexion of the neck with the muscles relaxed during anesthesia.[4, 17]

SUMMARY

In our experience with 239 cases of the shoulder, arm and hand syndrome, cervical disk was diagnosed in 132 cases and the scalenus anticus syndrome in 66 cases. Of the 132 cervical disks, 12 had laminectomy, and 28 of the scalenus group had section of the muscle. In over 80 per cent of these surgical procedures the results were excellent. It is our belief that in both these conditions, as well as other conditions producing shoulder, arm and hand pain, we should utilize conservative treatment as much as possible, and that the operative procedure should be used only as a last resort.

Many other syndromes have been described and have been mentioned in the first part of this paper. In our experience these syndromes are relatively rare. Overton's[9] study of the cervical spine and his two reports have been of definite benefit in giving us a better understanding of the pathological changes occurring in this area. The works of Russek, Walker and Spurling should also be mentioned because of their important contributions to this subject.

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EDITORIAL

IS PANCREATODUODENECTOMY FOR CARCINOMA OF THE AMPULLARY REGION JUSTIFIABLE AS A PALLIATIVE OPERATION?

Removal of carcinoma of various parts of the body has been accepted as good palliative treatment in selected patients known to be incurable. Life can be prolonged and made more tolerable in many cases. One may reasonably question the advisability of a palliative operation having the high mortality rate of pancreatoduodenectomy.

Broadly speaking, it might be said that all operations for carcinoma of the ampullary region have been palliative, since there is no known instance of a cure. While this is true, certainly pancreatoduodenectomies have been done with the intent to cure if possible. It is interesting and encouraging to note that I have found reports and received personal communications of 18 patients who have survived longer than five years after pancreatoduodenectomy. I know of no instance in which the radical operation has been done with the intent that it be palliative only.

As a palliative operation pancreatoduodenectomy deserves consideration. There is abundant evidence that the average length of life is longer after pancreatoduodenectomy than after the palliative by-passing operation to relieve jaundice. In my own experience with pancreatoduodenectomy, the average length of life has been 15.5 months, whereas the average survival time with the palliative by-passing operation has been 4.4 months. A careful analysis of published reports indicates that other surgeons have had similar experiences. It is worth emphasizing that all patients operated upon for carcinoma of the ampullary region, who are not suitable for the radical operation, should have a palliative by-passing operation when such is technically possible.

The high mortality rate of radical pancreatoduodenectomy probably has been a deterring factor in the use of this operation as a palliative procedure. It is true that the published mortality rates of pancreatoduodenectomy have ranged from 7.7 per cent to 45 per cent. It is also true that the mortality rates of the palliative by-passing operation have ranged from 7.4 per cent to 48 per cent. The high mortality rates in the latter group can, of course, be explained partially by the more serious condition of patients at the time of operation.

Child and his associates recently summarized a series of 1000 patients treated by palliative by-passing operations and found that the average length of life after operation was 7.5 months. By comparison these authors reported that the average survival time of 13 of their patients, treated by pancreatoduodenectomy, was 15 months. This average survival time compares favorably with the survival time of other malignancies treated by palliative operations.

The operative mortality rate of pancreatoduodenectomy is being lowered.

Some authors have reported it to be below 10 per cent. This decrease in the operative mortality rate has undoubtedly been due to a better understanding of the physiologic problems involved; increased experience and technical skill; better preoperative and postoperative care; better anesthesia; and a more careful selection of patients suitable for the radical operation. There are two principles of technic which have reduced both the morbidity and the mortality rate. These are the anastomoses of the severed ends of the pancreatic duct and bile duct to the intestine. These anastomoses reduce to a minimum the leakage of pancreatic juice and bile.

In published reports the evidence seems clear that the mortality rate of radical pancreatoduodenectomy is being reduced; that the mortality rate of the radical operation compares favorably with the palliative by-passing operation; and that the average length of life is much longer after pancreatoduodenectomy than after the palliative by-passing operation. For these reasons palliative pancreatoduodenectomy may be considered a justifiable operation in well selected cases without distant metastases.

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